The Journal

of the

Louisiana State Medical Society

$4.00 Per Annum, 35c Per Copy
Vol. 113, No. 2

FEBRUARY

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

Little Appreciated Facts and Occasional Oversights in Electrolyte Therapy*

• The author outlines the causes for subnormal concentrations of sodium, potassium, and magnesium, and gives suggestions for therapy.

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THE widespread use of the flame photometer has brought more awareness of the problems of fluid and electrolyte therapy, but as with any clinical tool, it has also brought new difficulties into the management of patients. The results of the chemical determinations often do not seem to correlate with the clinical picture, and many times ill-advised therapy has been prescribed on the basis of serum concentration alone.

Low serum concentrations do not always signify ion deficits, and in this paper the main causes for subnormal concentrations will be outlined, with a few suggestions for therapy. This discussion will be limited to the three major cations of the body; sodium, potassium, and magnesium. In spite of the title of this paper, no statements made here can be called facts, but are only theories, as knowledge of the basic physiology of electrolytes is only in its infancy.

To begin with, a few elemental premises need to be reviewed. First, disorders of electrolytes are rarely diseases in themselves, but are only manifestations thereof, and consequently, the main therapy must be directed at the primary pathology. This may seem obvious; however, it is often misleading. Patients in diabetic acidosis may temporarily respond very satisfactorily to intravenous saline, but they need insulin to get well; the patient with small bowel obstruction primarily needs a surgeon and not a fluid therapist. Furthermore, disorders of electrolytes often cannot be fully corrected until the primary disease is eradicated. It is often fruitless to try to restore a patient into perfect balance before corrective surgery is performed; it is only necessary that a start be made in the right direction, and in acute situations, this can usually be done in a matter of hours, rather than days.

Very little will be mentioned about clinical patterns, but it is wise to remember that symptoms and signs vary more with

* Presented at the Annual Meeting of the Surgical Association of Louisiana, Sheraton-Charles Hotel, New Orleans, Louisiana, November 20, 1960.

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the rapidity of change than with the absolute amount, and over a long period of time, large deficits are often well tolerated. Another salient point is that the normal route of electrolyte absorption is from the gastrointestinal tract. Consequently, as soon as possible, this route should be restored to full function and all parenteral fluids discontinued.

Sodium

The first ion that we will discuss is sodium. As can be seen in Figure 1, it

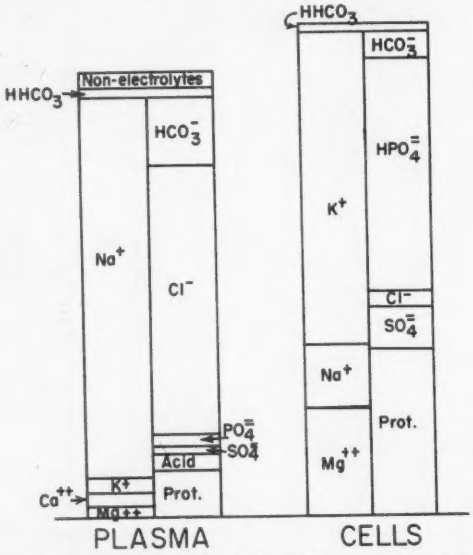

Figure 1.—Normal electrolyte concentration in plasma and cells (adapted from Bland).[5]

makes up almost all of the cation in the plasma and extracellular fluid. The tonicity or osmotic strength of the extracellular fluid is almost entirely dependent upon it. In other words, when the sodium is low the serum is hypotonic. Since all body fluids are in osmotic equilibrium, the extracellular and the intracellular fluids are also hypotonic.

There is one exception to this. If the nonelectrolytes are elevated, they can contribute to the osmotic strength of the plasma and in that case a low serum sodium does not represent a sodium deficit. These nonelectrolytes include glucose, lactate, pyruvate, bilirubin and fat, although ordinarily only a high serum fat will depress the serum sodium. Urea cannot be included as it is so freely diffusible that it does not affect osmotic relationships. Thus if the serum is lipemic, the sodium concentration may be low.[1] So we see the first condition in which a low serum sodium is not in itself pathologic and should not be treated.

But there are situations in which sodium therapy is needed. All body fluids contain sodium. When these fluids are a abnormally lost, sodium is lost, too. In general, these fluids are all secretions of the gastrointestinal tract and are all almost isotonic with plasma.[2] These intestinal juices can be lost from vomiting, diarrhea, fistulas, or occultly into the obstructed bowel. Since these solutions are isotonic, their loss is not initially reflected in any change of concentration of the serum sodium. This is analogous to an acute hemorrhage in which the hematocrit remains the same until dilution can occur. Thus, a patient can have a large and even life-threatening loss of fluid and still have normal serum electrolytes. In this early stage, therapy should be with isotonic solutions such as saline or Ringer's, or one of the newer solutions that attempt to correspond to the fluid lost.

In time, however, the body seeks to maintain the extracellular volume at the expense of concentration. This volume is necessary for proper cardiac action and blood flow. To do this, the kidneys conserve all available water and the electrolytes become diluted. Where does this water come from? The most common source is from administered water, either drunk by the patient or given parenterally as 5 per cent glucose in distilled water. Besides, there is another source of water, that is, from the catabolic oxidation of the body's own tissues. As this occurs, it produces electrolyte free water which dilutes the serum, rendering it hypotonic. The effect does not end here. Since water is freely permeable to cell membranes and the cells are then relatively hypertonic, water goes into the cells, causing them to swell, and thus the whole body becomes hypotonic. (Figure 2.) As will be dis-

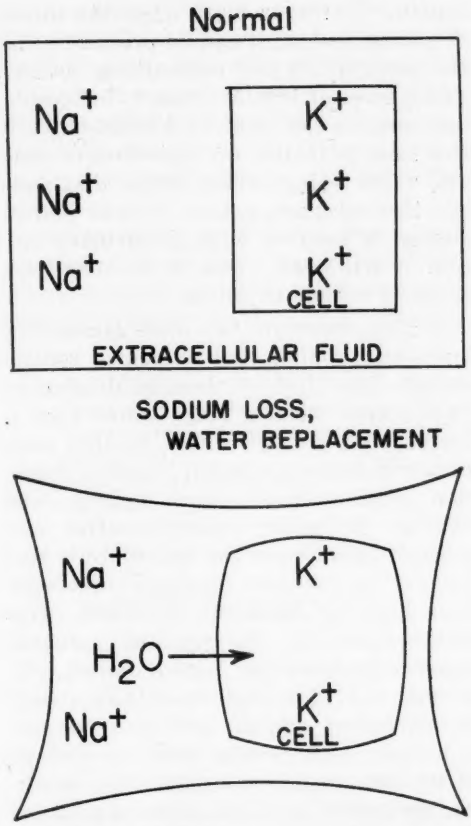

Figure 2.—Diagrammatic representation of total body water showing intra- and extracellular components. The lower figure shows cellular swelling and contracted extracellular volume occurring as a result of sodium loss and water replacement.

cussed later, potassium is also lost and this contributes to the hypotonicity.[3]

Therapy in this situation thus depends on two factors; the degree of hypotonicity, that is, the serum sodium; and the volume of the extracellular space, which may be normal, increased or markedly decreased, depending on the amount of water retained. The important thing is the amount of sodium which has actually been lost, the true sodium deficit, but present clinical methods cannot give us that figure.

Many authors advocate giving enough sodium to restore normal tonicity to the total body water, which is about 60 per cent of body weight. Their reasoning is as follows: When hypertonic sodium is given it raises the tonicity of the serum and the extracellular water. This draws water out of the cells and this water then serves to lower the concentration of sodium in the extracellular space. (Figure 3.)

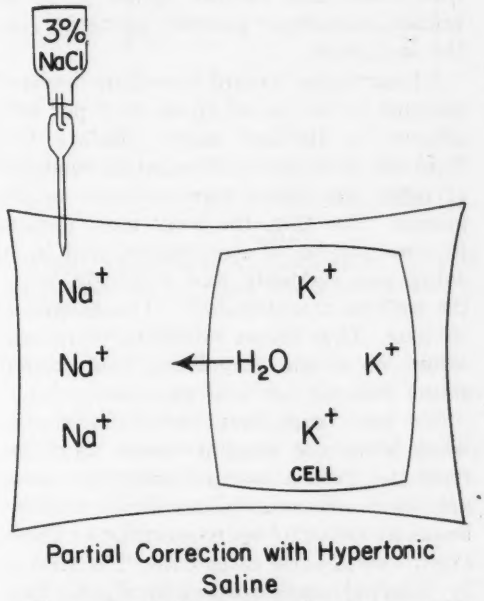

Figure 3.—As hypertonic saline is given intravenously, water is drawn from the cells to restore the extracellular volume toward normal.

By this reasoning a 60 kg. man with a serum sodium of 120 (instead of a normal of 140) would have a deficit of 20 mEq. per liter times 36 liters of body water, or 720 mEq. sodium. This is the amount of sodium in 1400 cc. of 3 per cent saline. But such large amounts are rarely needed. This reasoning would be correct if the total body water were constant, but it is not. As water is drawn out of the cells it expands the extracellular volume. As soon as this volume approaches normal, further water drawn from the cells is excreted in the urine.[3] Thus if the extracellular volume were normal to begin with, only about one-third as much sodium would be needed, and as stated before, there is as yet no practical method of measuring the extracellular volume. In therapy then, the answer is moderation. Continuing losses should be replaced with isotonic solutions, but for the hypotonicity hypertonic

saline should be given, but in relatively small amounts. Saline, 3 per cent, 300 to 500 cc. should be administered, and the body given twelve to twenty-four hours to equilibrate. Repeat serum studies are then made and further saline given as needed, as soon as possible, preferably by the oral route.

Of particular hazard in sodium depleted patients is the use of clyses of 5 per cent glucose in distilled water. Before this fluid can be absorbed, it must be rendered of equal electrolyte concentration as the plasma. For this, the body must deplete its own plasma of electrolytes, and in so doing can suddenly and seriously lower the sodium concentration. The hazard is obvious. Thus clyses should be of normal saline, or at the very least, half normal saline with 2.5 per cent glucose.

We have seen that sodium is needed when abnormal amounts have been lost from the gastrointestinal tract, but there are other abnormal losses beside obvious losses in sweat or by paracentesis. These are in two general categories. The first is by internal sequestration of fluid. This occurs locally in areas of trauma or inflammation, as in burns, or into the abdomen in peritonitis and pancreatitis. It also occurs in any area with acute venous obstruction. The second source of sodium loss is through the urine. Normal kidneys preserve sodium very well and can lower its excretion to virtually zero, but in certain disease states this ability is lost. In osmotic diureses, such as diabetes mellitus, large amounts of sodium are carried out before reabsorption can occur. In metabolic acidosis, sodium is used as a base to be excreted with the acid. In adrenal insufficiency, the kidneys lose the aldosterone effect of sodium reabsorption. Finally, in the even rarer disease of salt losing nephritis, the kidney has lost the ability to selectively save sodium. These latter causes must be ruled out.

If the serum sodium is low from extrarenal loss, the loss must be great and the source of loss should be apparent from the history and physical examination. Lacking that, the urine should be analyzed for sodium. If this is high, when the intake of sodium is low, it can be presumed that the kidneys are not reabsorbing sodium. Small doses of DOCA (desoxy-corticosterone acetate) can be given for one day.[8] If this fails to inhibit the excretion of sodium, a salt losing kidney can be diagnosed and then adequate sodium must be administered to keep up with the urinary output. Fortunately, this is a very rare cause of sodium depletion.

Finally, there are two more causes of a low serum sodium, but without a sodium deficit. The first of these is dilution.[1, 3] This occurs when a large water load is forced on a patient whose kidneys have impaired water excretion. Such a condition exists in shock and in organic renal disease. It occurs routinely after any severe trauma when the anti-diuretic hormone of the posterior pituitary is secreted. This hormone markedly decreases urine water excretion. Surgery and narcotics enhance its secretion. Such a patient with normal total body sodium suffers a drop in his serum sodium, and thus becomes hypotonic with cellular swelling and all of its hazards. Some hypotonicity is normal postoperatively, but large infusions of 5 per cent glucose in distilled water, frequent enemas or large oral intakes can intensify this condition. It can be recognized by a lethargy progressing to semicoma. Treatment is simple water restriction. If water loading is acute, convulsions can occur from cerebral edema. Then, hypertonic saline is needed; 300 cc. of 3 per cent will usually suffice, but remember, the body does not need sodium, it simply has too much water.

The last cause of a low serum sodium and the one least often recognized can be called asymptomatic hyponatremia. It occurs in any chronic illness and in convalescence from any severe trauma. The low serum sodium (generally 125 to 130 mEq./liter) reflects hypotonicity of the entire body, but there is no sodium deficit. Administered sodium is excreted quantitatively in the urine, and except very temporarily, the serum level cannot be raised by infusions of sodium chloride,

no matter of what concentration or amount. The cause is apparently an energy deficit and the only cure is improvement in the primary condition. To give these patients large amounts of sodium is especially hazardous, as it may lead to congestive heart failure and pulmonary edema.

In summary, we see that there are a number of causes for a low serum sodium, many of which do not require sodium for therapy. (Table 1) Usually, if there has

TABLE 1
CAUSES OF LOW SERUM SODIUM

1. Increase in non-electrolytes
2. Sequestration of sodium containing fluid within body
3. Loss of sodium from body
   a. Gastro-intestinal tract
   b. Urine
   c. Sweat, spinal fluid, paracentesis, etc.
4. Dilution
5. Asymptomatic Hyponatremia

been sodium loss, the source of loss is obvious and the treatment will be as outlined. However, there will be cases in which the need for sodium is in doubt. In this group it is safe to infuse 300 cc. of 3 per cent saline and observe the results. If no improvement occurs, be cognizant that it may either be dilution, or more likely, an asymptomatic hyponatremia.

Potassium

The second ion to be discussed is potassium, which is chiefly an intracellular one. Potassium is present in all body fluids and is excreted in large amounts in the urine, usually 30 to 60 milliquivalents a day, a loss which usually continues in spite of a body deficit. In any disease state in which there is either an abnormal loss of body fluids or an absence of potassium intake, a deficiency state can occur. As potassium is lost from the cells, it is replaced by sodium and hydrogen ions in the ratio of two sodium to one hydrogen.[9] (Figure 4.)

Since these hydrogen ions are supplied by the extracellular fluid, it is obvious that the extracellular fluid becomes more alkaline. This is independent of any renal mechanism and is reversible by the administration of potassium. A similar transfer mechanism occurs in the kidneys, and in the presence of an alkalosis, the kidney tries to save hydrogen ions, and in its place, excretes sodium and potassium. Thus, potassium deficiency leads to alkalosis and alkalosis leads to potassium deficiency. Neither can be corrected without the administration of potassium.

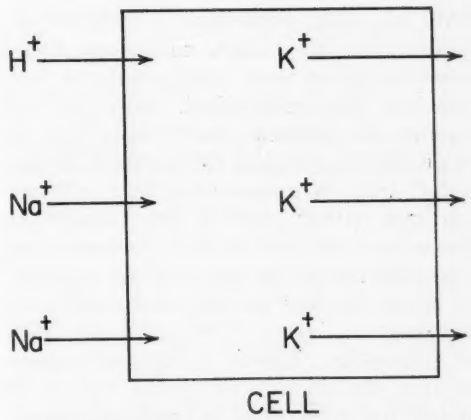

Figure 4.—Cellular potassium loss is replaced by sodium and hydrogen ions from the extracellular fluid.

Potassium deficiency is particularly evident in patients with high intestinal obstruction such as an obstructed duodenal ulcer. In vomiting acid gastric juice, hydrogen ions, potassium, chloride and sodium are lost. When these patients are first seen, their serum electrolytes usually show a low sodium, a low chloride, a high CO_2, and a low or normal potassium. Administration of saline may improve the patient considerably, but the alkalosis will persist until potassium is given.

How much potassium to give? Again it is impossible to know the true deficit, but there are guide posts. The maximum amount ever lost in any condition is about 17 mEq. per kilogram, or about 1,000 mEq. in a 60 kilogram man. Usually the need is one-third to one-half this amount. This of course should not be administered in any one day. The maximum safe dosage is 3 mEq. per kilogram per day, with no more than 60 mEq. in any one liter of fluid. The danger of giving more than this of course, is of raising the serum

level too high, with toxic effects on the heart. For this reason, potassium should never be given until adequate urine output has been established. Once positive balance is restored, many days may be required for complete replacement. Where a deficiency is compounded by a continuing loss, either through the kidneys or from the intestinal tract, it is important that adequate potassium be given each day to cover the loss as well as to make up the original deficit. Ordinarily 40 mEq. of potassium chloride a day will cover urinary excretion; 10-15 mEq. should be added for each liter of gastrointestinal fluid lost from the body.

Magnesium

The last ion to be discussed is magnesium. Only recently has a deficiency been recognized in man.[10-14] Magnesium is found chiefly intracellularly, half of it being present in bone. The normal daily intake is 200 to 300 mgms. Its absorption from the gastrointestinal tract is under no known control. Urinary excretion is temporarily enhanced by aldosterone and parathyroid hormone, but this effect does not continue.

Magnesium has both a local and a central effect on the central nervous system. Locally, a decreased magnesium level causes an increase in nerve conduction and an increase in transmission of impulses at the motor end plates. Centrally, a magnesium deficiency produces excitability and irritability. The normal serum magnesium is 1.8 to 2.1 mEq. per liter. Low levels are seen in chronic nephritis, congestive heart failure which has been treated by ammonium chloride and mercurial diuretics, and in acute pancreatitis, where like calcium, magnesium is deposited with fatty acids as soaps. In none of these conditions, however, has magnesium deficiency been shown to play a part in the pathologic process. Low serum magnesium levels are often seen in chronic alcoholics, but there is probably no relationship between this and the development of delerium tremens.

There is, however, a syndrome of magnesium deficiency. It occurs where there is a prolonged absence of oral intake coupled with an abnormal loss, either through the gastrointestinal tract or the urine. The signs and symptoms are those of irritability progressing to tetany and are indistinguishable from those of calcium deficiency, except that there will be a normal serum calcium and a low serum magnesium. However, the determination of serum magnesium is still difficult, and only a few laboratories render this service. If the syndrome is suspected, magnesium therapy should be begun. One word of caution: both a low serum potassium and a low serum calcium antagonize the effects of a low magnesium. This is of clinical importance. First, in a combined potassium and magnesium deficiency, the magnesium deficiency may not manifest itself until the potassium level is restored. Second, if calcium is given intravenously to patients in magnesium tetany, it will only aggravate the condition. Magnesium therapy should be given as a fifty per cent solution $MgSO_4$ intramuscularly. Four ccs. can be given every six hours for one or two days. It can be given intravenously, but high serum levels of magnesium are sedative and can produce coma. Since this is an intracellular ion, improvement is slow, generally requiring twelve to twenty-four hours before any change is seen in the clinical state.

Summary

We have briefly covered some abnormalities of the three major cations of the body. Serum concentrations of these ions are often useful in gauging their clinical need, but several examples have been given in which a low serum content is a result of causes other than a body deficiency of the ion. Proper therapy thus demands a complete evaluation of the patient. In doubtful cases, conservatism should be the rule.

Finally, a word of solace to all practitioners of the electrolyte art. There will be some cases which absolutely defy all rational explanation, but there is one hope; if the basic disease can be eradicated and

oral alimentation restored, there will shortly be no electrolyte problem.

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To Our Readers—A New Proposition

The Managers of this Journal announce a new departure at the end of the present number which closes the volume. We have long recognized the desirability of conducting it on strict business principles, but have so far felt unable to break off from the traditions and habits of the past. The subscription price of the Journal, once more and never less than five dollars a year, was expected to cover the risks of an indefinite credit. Changes from a bi-monthly to a monthly and from 80 pages in number to 100, do not seem to answer the wants of physicians in the Southwest. Evidently they require a cheaper journal, like their brethren in other parts of the country, and we shall attempt to answer the demand. We therefore propose to reduce the subscription price to three dollars a year, from July 1, 1881; but as this moderate rate allows no margin for risks, we are under the necessity of insisting strictly on payment in advance.

New Orleans M. & S. J. 8:1183, (June) 1881.

Postpartal Pneumoperitoneum

• The author gives an account of a case in his own experience caused by the knee-chest position exercise and cites others from the literature.

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THE title of this paper is so given to focus attention on pneumoperitoneum occurring in the postpartum period unrelated to a primary diseased state.

Pneumoperitoneum is always the result of an induced procedure or a complication of a previously existing disease. Spensley, Nelson, and Childs [10] made a fairly comprehensive review of the existing literature and listed the following causes for pneumoperitoneum:

1. Perforated benign ulcerations of the stomach and duodenum.
2. Perforated malignancies anywhere in the gastrointestinal tract.
3. Perforated ulcerations of the bowel due to (a) Typhoid (b) Tuberculosis (c) Regional enteritis (d) Ulcerative colitis and (e) Foreign body.
4. Ruptured appendix.
5. Ruptured Meckel's diverticulum.
6. Ruptured diverticulum of the colon.
7. Following laparotomy.
8. Following gastroscopy.
9. Following the Rubin test.
10. Following the knee-chest position exercises in the postpartum female.
11. Following use of a douche.
12. Trauma to the rectum or sigmoid with an enema tube or other instrument.
13. Blunt trauma to the abdomen.
14. Pneumatosis cystoides intestinalis.
15. Peforation of the bowel due to an obstruction.
16. Idiopathic perforation of the bowel.
17. Idiopathic perforation of the stomach.
18. Following therapeutic pneumothorax.
19. Intraperitoneal abscess with gas forming organisms.

They also mention that pneumoperitoneum has been associated with tonsillectomy, pharyngeal diverticulectomy, pulmonary bullae and pneumonia with the causal relationship underlying not being clear. It is interesting to note that in their list they include (Number 10) pneumoperitoneum occurring in the postpartum female.

Case Report

The case as reported here and those in the prior literature appear to have occurred by an unusual mechanism, that is, by an unexpected passage of air into the abdominal cavity by way of the female genital tract.

The patient, a 33 year old housewife, was seen at home about 11:30 a.m. on December 15, 1959. About 10:00 a.m. she developed a severe epigastric pain which became generalized across the upper abdomen. The pain radiated into the right chest, shoulder, right side of her neck and down into the right arm. The pain was sharp and aggravated by taking a deep breath, changing positions, and swallowing. She had no fever or cough. She had noticed no previous abdominal pain. At this time she was three weeks postpartum and had been taking knee-chest exercises since the second or third postpartal day. The onset of her symptoms occurred approximately one hour after taking the morning's exercises. (She was aware that air had entered the vagina during her exercises. She mentioned this later after the diagnosis had been established.)

Her past health had not been remarkable. She had been periodically bothered by sinusitis. Three previous pregnancies had been uneventful.

Physical examination showed a well nourished, healthy but apprehensive young lady. Her temperature was 98.4°F. Her blood pressure was 110/70 mm. of mercury. The pulse rate was 90 per minute. Her physical examination was entirely normal except for tenderness to pressure over the right abdomen and absence of hepatic dullness to percussion. Pelvic examination was not performed.

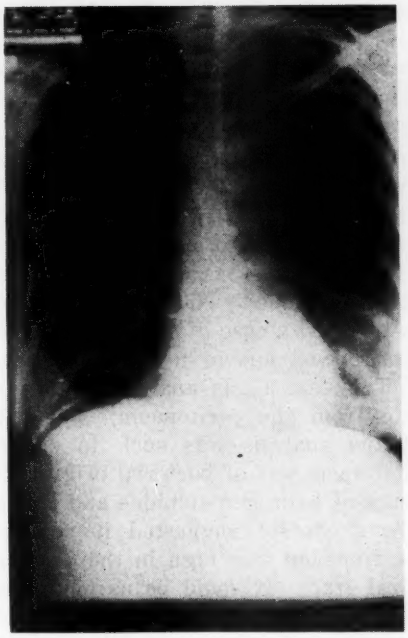

Figure 1.—A moderate amount of subdiaphragmmatic air is present. This was taken approximately four hours after the onset of symptoms.

In view of the patient's apparent good health, a tentative diagnosis of a displacement of the intestine or colon over the dome of the liver was made. She was sent to the hospital, ambulatory, where a roentgenogram (Figure 1) of the lower chest and upper abdomen was made. This showed air under the diaphragm establishing the diagnosis of pneumoperitoneum.

Her course was uneventful, the pain on changes in position being noted but gradually decreasing until it disappeared within the next thirty-four to thirty-six hours.

Comment

The general healthy condition of this patient pointed to a benign etiology for the pneumoperitoneum. This is not so when, as most usually seen, it is due to a perforation of the gastrointestinal tract. The benign clinical picture has been well pointed out in the discussion of a similar case by Conn and LaFon.[1] Their case was that of a young lady five weeks postpartum. The pneumoperitoneum in this case recurred two months later while pelvic examination was being performed in the knee-chest position.

The cases of Bean and Garrett,[2] Dodek and Friedman,[3] Lozman and Newman,[15] were also similar. In all cases the patient was a healthy, postpartal young lady. Upper abdominal pain and thoracic pain related to respiration and changes in position developed after assuming the knee-chest position. Loss of hepatic dullness to percussion was the pertinent physical finding in the case reported and in 2 others. In 1 case no mention was made of the physical findings and in the other the only noted abnormal finding was diminished breath sounds over the right lung field posteriorly. Roentograms of the chest established the presence of a pneumoperitoneum in all.

The mechanism responsible for the passage of air into the abdominal cavity by the way of the genital tract is not completely clear. It is suggested by the cases reviewed that two factors are of the utmost importance, one being a more patent genital tract allowing air to more easily pass through the vagina, uterus, and fallopian tubes. It would seem probable that this patency would exist toward the end of the sixth week, this being the time required for involution of the uterus.[7] In performing the Rubin's test in the normal female, it has been found that from 70 to 100 mms. of mercury pressure is required to force air through the normal oviducts.[4] In this respect cases of pneumoperitoneum have been reported due to the passage of douching fluid and air under pressure through the genital tract.[3, 6, 10, 14]

The second and probably the most important factor is the knee-chest exercise. It has been known that upon assuming this position with the chest down, the vagina will balloon out and fill with air.[4] It then seems reasonable as mentioned by Redfield et al[9] that when the patient descends from this position the labia close and with collapse of the uterus and vagina air is forced out into the oviducts and escapes into the abdominal cavity if it cannot escape through the vaginal orifice below. The descent of the viscera toward the chest probably creates a decreased

intra-abdominal pressure enhancing the passage of air. Garland [8] mentioned a patient who developed a pneumoperitoneum on two occasions after vigorous knee-chest exercises on the advice of a cultist. The recurrence in the case reported by Conn and LaFon was in conjunction with a pelvic examination performed in the knee-chest position. It is probable that pneumoperitoneum as a consequence of knee-chest exercises in the postpartum period occurs more frequently than the reports in the literature would suggest.

Schneewind [11] reported a case of a 25 year old woman on whom an exploratory laparotomy was performed for an unexplained pneumoperitoneum. Nothing was found except a moderate mesenteric adenopathy. In the history it was mentioned that she had previously been an obstetrical patient. In his discussion he mentioned sources, such as, (1) escape of gas from minute perforations of the stomach or upper intestinal tract, (2) from the chest via a lymphatic hiatus and due perhaps to rupture of an emphysematous bleb, and (3) vaginal through the fallopian tubes following douching. It was not mentioned whether this patient had been performing knee-chest exercises or how long prior to the pneumoperitoneum she had been an obstetrical patient.

That the use of the knee-chest exercises in the postpartum period is not always innocuous is attested by the reports of air embolism with death occurring in the postpartum period. Such cases have been reported by Redfield et al [9] who reviewed 2 cases from the literature and reported two cases of their own. These cases occurred from the seventh to the eleventh postpartum day.

Karondy et al [16] in their discussion of fatal air embolism mentioned a report of Brodsky and Greenstein [17] of fatal air embolism following the knee-chest exercises. Waldrop [13] reviewed 4 previously reported cases of fatal air embolism following the knee-chest exercises. Two of these were the cases reported by Redfield et al. The likely route by which air was introduced was by way of the genital tract into the subinvoluted uterus then through the uterine veins. In these cases it seemed apparent that the knee-chest exercises produced the pressure required for the entrance of air in this manner.

In the cases reviewed of the mechanism by which air entered the abdominal cavity that it was atmospheric air has only been deduced by the histories and examinations of the patients. A more scientific approach was made by Nanson and Dragon [12] who reported a case of spontaneous pneumoperitoneum due to jejunal diverticulosis. They did a gas analysis on the aspirate from the peritoneum. They felt that the analysis was such to suggest that the gas was of bacterial origin. The absence of hydrogen sulphide and the low methane content suggested it not only came from but was high in the gastrointestinal tract. It could be assumed that if gas analysis had been made from the peritoneal air in the postpartal cases that it should have been of atmospheric content.

Summary

1. A case of pneumoperitoneum in the postpartum period is reported. Other cases of pneumoperitoneum of genital origin have been reviewed.

2. The mechanism of the pneumoperitoneum as related to the knee-chest position and a subinvoluted uterus with the dangers inherent are mentioned.

3. A suggestion is made that knee-chest exercises be discontinued until the time required for involution of the uterus has passed.

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Soluble Compressed Pellets for Hypodermic Use

We have received from John Wyeth & Brother a case containing specimens of these pellets in small vials, variously composed of morphine, and morphine with atropine, rendered more soluble by combination with sodium sulphate. Each pellet is intended for a dose, to be dissolved in a suitable quantity of water. A convenient method is to break a pellet into fragments in a spoon; then take up a few minims of water with the hypodermic syringe and throw it into the spoon to dissolve the dose, when ready for use. The advantage of this plan is the avoidance of the penicillium, which is sure to form, sooner or later, in the solutions kept for use. A few drops of chloroform in the solution will, however, greatly retard the appearance of this vegetation; but, unless one has quite frequent occasion to use the hypodermic syringe, he will probably find the pellets an improvement on the readymade solution.

New Orleans M. & S. J. 8:1187 (June) 1881.

Primary Common Bile Duct Stones*

• The value of intravenous cholangiography using cholagrafin methylglucamine to demonstrate choledochal stones is well illustrated by two case reports, as well as the fact that stones may occur in any part of the ductal system without having first originated in the gallbladder and later moved into the ducts.

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COMMON bile duct stones occur most often in association with stones in the gallbladder, and when they occur as a post-cholecystectomy complication it is usually within a few months after operation. Until recent years whenever a patient said that the gallbladder had been removed *many* years ago, one generally dismissed the biliary system as a source of upper abdominal or chest pain and concentrated his efforts on other organs or organ systems. However, since the advent of intravenous cholangiography with Cholagrafin more of the cases of late post-cholecystectomy comman duct stones are being accurately diagnosed.

This paper was prompted by two such cases that came to my attention in 1959. One had had a cholecystectomy for cholelithiasis twenty-five years previously; the other thirty years.

Case Reports

The first case is that of an obese 50 year old white woman, the mother of two children, who had a cholecystectomy for cholelithiasis in 1934. For twenty-three years she enjoyed good health insofar as her digestive tract was concerned. In the twenty-fourth year she complained of vague upper abdominal and precordial pains, which were at first thought to be of cardiac origin because her gallbladder was out.

She was obese, a known hypertensive, and sometimes got quick relief with nitroglycerin. However, in the ensuing two years she went through periods of complete freedom from discomfort alternating with pains that were variably either in her upper abdomen, precordium, or back. On one occasion she was hospitalized for suspected coronary thrombosis.

Complete laboratory, x-ray, and repeated electrocardiographic studies were all normal. At no time was she ever jaundiced, clinically or chemically.

Finally, in March 1959, intravenous cholangiography was done using Cholagrafin methylglucamine. The widely dilated common duct was adequately visualized but because of the overlay of intestinal contents one could not be certain of the nonopaque stones which were brought out admirably, using body section radiography.

Accordingly on April 29, 1959, exploration of the common bile duct was done, and multiple large stones which were adherent to the wall of the dilated and loculated common duct, and a single large stone adherent to a cystic duct remnant, were removed. Bake's dilators were easily passed and the surgeon felt that there was no evidence of obstruction at the sphincter of Oddi. A T-tube was left in the common duct and the patient recovered uneventfully and went home on the eighth postoperative day. Three weeks later the T-tube came out inadvertently one night.

The patient got along fine, completely free from symptoms, on an unrestricted diet, until January, 1960, at which time she was again seized with what she stated was the worst pain she ever had—right upper quadrant pain radiating to the back and precordium. This pain was completely relieved by 200 milligrams of Demerol orally.

Another electrocardiograph was normal and intravenous cholangiography did not reveal any stones. She probably passed a small reformed stone and has remained well to the present time.

The second case is that of a white man aged 61, whose first trouble began in 1921, when he had a cholecystostomy, his gallbladder being emptied of multiple stones. At that time he had fever, jaundice, distention, vomiting, and was said to be quite toxic. He gradually recovered and remained well until 1929 when he had a recurrence of cholecystic symptoms, which culminated in a cholecystectomy in the fall of that year.

After this he stayed well, with the exception of occasional attacks of gout, until 1955. At

* Presented at the Eightieth Annual Meeting of the Louisiana State Medical Society, in Baton Rouge, May 4, 1960.

that time, he began to have intermittent attacks of some upper abdominal pain which sometimes radiated into the precordium and back. These attacks were often associated with mild degrees of jaundice, and occasional fever, with or without chills, and sometimes the pain was severe enough to require hypodermic medication. At other times, severe pain of short duration would come on suddenly and then leave just as suddenly. He traveled widely, saw many physicians, and had various diagnoses made.

In April 1959, about one week after returning from a vacation with a severe chest cold, he was first seen at home with a recurrence of severe upper abdominal and chest pain, jaundice, chills and fever 103°F. He was hospitalized with tentative diagnosis of common bile duct stones and acute cholangitis.

Chest x-ray revealed pneumonitis and atelectasis at the right base which may have been the cause of fever with this attack.

At this time he had a leukocytosis of 15,000 with 96 per cent neutrophiles, serum bilirubin of 7.7 and icterus index of 32.5.

He responded well to penicillin and streptomycin, and infusions, and was fever free in five days. On the eighth hospital day he was suddenly seized with an acute attack of gout, from which he likewise recovered.

He convalesced at home until he was symptom free and chemically normal, so that in May 1959, intravenous cholangiography was carried out. The common duct was greatly dilated and showed large radiolucent stones. Body section radiography was valuable in clarifying the existence of these stones. In this case a postoperative T-tube cholangiogram showed the ducts to be free of stones.

In July 1959, exploration of his common bile duct revealed it to be greatly dilated, harboring three large and several small stones that were free in the duct, and also a large stone in the remnant of the cystic duct. Bake's dilators were likewise passed with ease in this case and the surgeon felt that there was no obstruction at the sphincter of Oddi.

His postoperative course was also uneventful, and he went home on the eighth postoperative day after having a T-tube cholangiogram which showed the common and hepatic ducts to be free of stones. In this case the T-tube was removed on the twelfth postoperative day and the patient has remained well to date.

Comments and Discussion

From the standpoint of pathophysiologic and clinical manifestations, the entire subject of biliary tract stones is a fascinating and often perplexing one that lends itself readily to almost endless discussion and speculation. It is therefore the purpose of this presentation to do no more than point up some of the interesting and salient features of the two unusual cases here presented, and to bring to your attention the value in such cases of the relatively new diagnostic tool, intravenous cholangiography with Cholagrafin methylglucamine.

First of all, the stones in these two cases are called "primary stones" to indicate that stones can and do occur in any part of the ductal system without having first originated in the gallbladder and later moved into the ducts. Such stones are not common, but nevertheless, it is well to bear in mind that this does happen, and that the removal of the gallbladder does not necessarily mean the end of gall stones even if the ductal system was free of them at the time of operation. Almost everything written about choledochal stones refers to them as retained, residual, overlooked, or reformed stones, the latter implying that a nidus was left at the time of surgery. Likewise, almost all such stones occur relatively soon after cholecystectomy, generally a matter of months and *not* years. Thus, it seems reasonable to say that the stones in these two cases, manifesting themselves twenty-three and twenty-five years after operation, originated in the respective common ducts which in effect had become common duct "gallbladders". It might be added that there is some experimental evidence to show that this can occur.

The wide variability in clinical manifestations of common duct stones is also admirably brought out by these two cases. Actually, the only symptom common to both was pain, and even this was inconsistent. In the first case, the pain was vague upper abdominal, not clearly right upper quadrant, sometimes precordial, generally not present at more than one location at a time; usually no more than a deepseated awareness of discomfort; sometimes relieved by nitroglycerin; and only severe enough on one occasion in two years to require a hypodermic for relief.

By contrast, the pain in the other case varied over a period of five years from deep-seated discomfort to severe excruciating pain which came on suddenly and sometimes disappeared quickly, while at other times it lasted for hours with severe exacerbations and required hypodermic relief. When first seen this patient actually described rather typical common bile duct distribution of pain, i.e., upper abdominal mainly right upper quadrant, radiating into the back and into the precordial area, depicting the pain that occurs most often with a stone in the ampulla of Vater and concomitant contraction of the sphincter of Oddi.

In regard to jaundice, which occurs in about 75 per cent of the cases of common duct stone, one of these was never jaundiced clinically or chemically, while the other was frequently jaundiced. Occasional fever with or without chills occurred in the jaundiced patient only, and probably represented instances of either cholangitis (choledochitis) or Charcot's intermittent hepatic fever.

These variations in clinical manifestations are explained well by the underlying pathology demonstrated at surgery. In the patient that was never jaundiced, the large stones were actually adhered to the wall of the common duct and were thus immobile, but allowed a free flow of bile on the non-adherent side of the stones. In the jaundiced case there were both large and small stones floating free in the duct, which readily explains the more severe and variable pain as well as the intermittent jaundice which was at times associated with chills and fever. Variable degrees of obstruction either by a ball-valve effect of a large stone, or by impingement and subsequent passage of smaller stones seems sufficient to explain these different manifestations.

Prior to seven years ago there was no consistently good way to prove the existence of stones like these, short of common duct exploration. As you all know by far the majority of stones are mixed non-opaque ones composed chiefly of cholesterol, and in the past generally were not well demonstrated by the oral administration of any dye. Thus the introduction of Cholagrafin into this country, in 1953, for the first time made it possible to satisfactorily visualize the biliary ducts by intravenous cholangiography. Cholagrafin sodium was first introduced, but was shortly supplanted by Cholagrafin methylglucamine which gives better visualization and practically no serious side reactions when properly used.

As you have seen in these two cases the use of body section radiography in conjunction with intravenous cholangiography, by eliminating the overlay of intestinal contents, is extremely helpful in making certain that the negative shadows seen in the duct are due to non-opaque stones.

About 85 per cent of the time the common duct is visualized with Cholagrafin. This has led to some very interesting findings regarding the size of the common duct in both the pre- and post-cholecystectomy state. The size of the normal duct has been variously placed between 5.5 and 7 mm. in diameter, and one eminent group states that normally the duct may vary from 2 to 14 mm. averaging 5.5. They contend that many of the ducts formerly considered dilated after cholecystectomy should be considered normal up to 14 mms. These figures furnish a good working basis at the present time, but it is probable that further studies on *different age groups,* using *modes* instead of *averages,* could come up with more nearly correct figures. Ducts as large as 30 mms. in diameter are reported in obstructed cases, and the ducts in the two cases reported here measured 22 and 25 mms. in their widest diameter.

Conclusion

These two remarkable cases have clearly indicated that stones may *originate* in the common bile duct decades after cholecystectomy, and should not be overlooked in the differential diagnosis of upper abdominal or chest pain. Intravenous cholangiography with Cholagrafin Methylglucamine in its present form offers a

safe and satisfactory means of clinching the diagnosis, and where indicated should be used in conjunction with body section radiography.

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The President's Case

The medical Gentlemen in charge of the President* seem to have quite abandoned hopes of his recovery. The civilized world joins in lamenting the tragic occurrence which may result in the death of the chief magistrate of a great nation. But, in connection with this event, our profession cannot hope to escape severe inquisition and criticism on account of its failure to secure a different issue. There is a feeling common to the laity, and one can scarcely declare that our own ranks are free from it, that competency in practice is inseparable from successful results. While in our reflecting moments we all understand how incorrect this method of reasoning is, it is yet true that we may fail in duty to ourselves and our colleagues in question, if we do not at all times, and under all circumstances, defend them from such assaults and prejudices as we know to be unjust.

One of the most embarrassing duties the medical attendants of the President have attempted to perform has been that of issuing frequent bulletins for the Associated Press. * * * The occurrences of the sick room—even those of the greatest moment to the physician, or patient, are very differently appreciated by the average newspaper reader. The surgeon is intensely gratified that his patient retains an enema until his stomach recovers its functions; but when he undertakes to recite the process to the outside crowd, they overwhelm him with ridicule.

* * *

We know nothing of his motives and have not been advised of anything to condemn in his conduct. But when Dr. Bliss informs a newspaper reporter of the occurrences between himself and Dr. Baxter, and adds that "Dr. Baxter will never be Surgeon General," it looks to us very much like evincing a disposition to suborn the prerogatives of his own position to work detriment to another.

New Orleans M. & S. J., 8:231 (September) 1881.

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* James A. Garfield

Diseases About The Major Salivary Glands; Differential Diagnosis

• This article deals largely with the diagnostic roentgen technique, sialography. This technique was recently modified by the author and co-workers at the University of Michigan Medical Center.

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THIS brief discussion of diseases of the parotid and submaxillary salivary glands will deal largely with differential diagnosis of some of the more common inflammatory and neoplastic conditions which may involve these structures.

The most important diagnostic aid in salivary disease in addition to an accurate history and complete physical examination, is the local evaluation. This must include palpation, determination of the type of material, if any, that can be expressed from Stensen's or Wharton's duct as well as probing. In the majority of cases, the technique of sialography should also be utilized.

Sialography is the process of demonstrating roentgenographically the duct system of the salivary glands after the injection of radiopaque media (Fig. 1). The important features of this technique are the use of a polyethylene catheter inserted into either Stenson's or Wharton's duct, a closed system of injection and physiologic reflex stimulation of the salivary gland to evacuate the contrast material by the patient's chewing a potent sialogogue such as a lemon slice.[1] This allows for roentgen examination of the filling phase and postevacuation filming of the emptying phase as well. Emptying films have revealed patterns which have been of assistance in differentiating normal and diseased conditions.[2, 3] The total amount of contrast material injected when the glands are diseased varies between 0.5 cc. and 1.5 cc. and rarely exceeds 2 cc. This method, termed physiological or secretory sialography will demonstrate dilation, obstruction, displacement and obliteration of salivary ducts as well as intraglandular inclusions such as calculi.[4]

Inflammatory Diseases of the Salivary Glands

The inflammatory diseases of the salivary glands may be divided into two general groups:

1. Chronic obstructive sialodochitis—abnormalities primarily in the main duct.
2. Chronic nonobstructive sialodochiectasis — abnormalities primarily in the peripheral ducts (sialectasis).

Chronic Obstructive Sialodochitis. The chronic obstructive inflammatory diseases of the major salivary glands are due primarily to stones and strictures. In effect this is a suppurative sialadenitis and is due to secondary infection subsequent to continued partial obstruction of the main ducts and to salivary flow.

Patients suffering with chronic obstructive sialadenitis are subject to acute exacerbations of infection characterized by swelling of the gland, pain, temperature and purulent discharge from the main duct orifice. When sialolithiasis is considered, the submandibular salivary gland claims 75 to 85 per cent of cases. Approximately 20 per cent of salivary gland calculi are nonopaque. In the case of the

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Permission has been granted by W. F. Prior Co., Hagerstown, Md. to use Figures 7, 8, 9 and 14 from Chapter 9 of Coates-Schenck-Miller Textbook of Otolaryngology, by I. M. Blatt, J. H. Maxwell, and J. E. Magielski.[4]

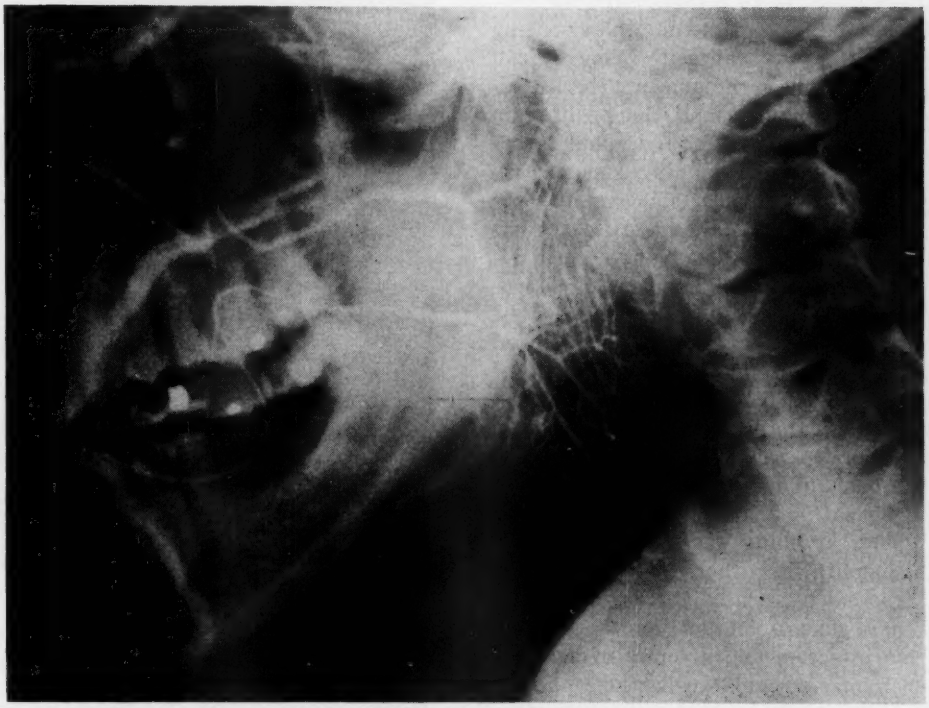

Figure 1.—Normal sialogram of the parotid glands. Filling phase of each gland was made consecutively.

Figure 2.—Sialogram of a submaxillary calculus. The filling phase (left) demonstrates a calculus near the hilum of the gland, lodged in the proximal dilated portion of Wharton's duct. The emptying phase (right) shows that upon stimulation, the salivary flow forces the stone into the narrower, distal portion of Wharton's duct. This poststimulation film demonstrates the ball-valve mechanism of the obstruction to salivary flow which is the basis of acute pain and swelling precipitated by eating.

small calculus (1 to 3 mm.) bidigital palpation in the floor of the mouth or probing the duct fails to localize it. Whether calculi are opaque or radiolucent, the knowledge of the number and the position in relation to the main duct and gland parenchyma will aid in planning surgical removal. In this regard, sialography is a useful diagnostic aid in identification as well as localization of salivary stones.

In Figure 2 the filling phase of sialography demonstrates a calculus near the hilum of the submandibular gland and lodged in the proximal dilated portion of Wharton's duct. The post-stimulation film demonstrates the ball-valve mechanism of obstruction to salivary flow which may be the basis of pain and swelling precipitated by eating.

In long standing cases of sialadenitis with sialolithiasis, there may be loss of the arboreal pattern of the normal gland. The main duct is dilated; and because of chronic infection and atrophy of salivary parenchyma, irregular collections of contrast material suggest abscess formation within the gland (Fig. 3). Occasionally

Figure 3.—Sialogram of a submaxillary calculus associated with chronic sialadenitis. The arboreal pattern of the peripheral duct system is obliterated. The main duct is dilated with irregular collections of contrast material which suggests abscess formation within the gland.

calculi may erode the wall of the main duct and become lodged in the gland substance. This results in production of chronic infectious granulomas which in themselves cause continued suppuration and further duct obstruction. Total parotidectomy with a complete anatomical dissection of the facial nerve is frequently the only suitable treatment for this form of chronic gland suppuration (Fig. 4).

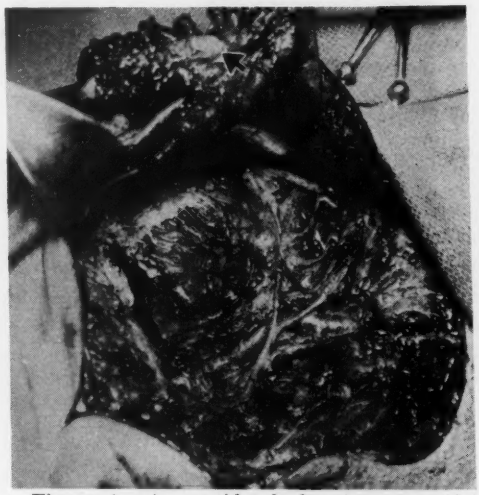

Figure 4.—A parotid calculus (arrow) which eroded the wall of Stensen's duct and lodged in the gland substance. This resulted in a chronic infectious granuloma which required parotidectomy.

Strictures, as they involve the salivary glands, are obstructive phenomena invariably associated with Stensen's duct and are a frequent cause of recurrent parotid swelling. This condition appears definitely associated with alteration in the mechanism of mastication in a manner which does not sustain the normal chewing habit. This type of obstructive sialodochitis occurs in the naturally dentitioned patient and in those individuals with partial or complete dentures which are ill fitting.[5]

As a result of the altered mastication mechanism, strictures occur at the parotid papilla which is subjected to repeated trauma, inflammation, fibrous tissue proliferation and scar tissue cicatrization. This interferes with normal salivary flow and the ensuing pressure behind the stricture results in dilation of the gland duct. It is not long before secondary bacterial infection supervenes with diffuse inflammatory reaction along the bucco-masseteric course of Stensen's duct and with

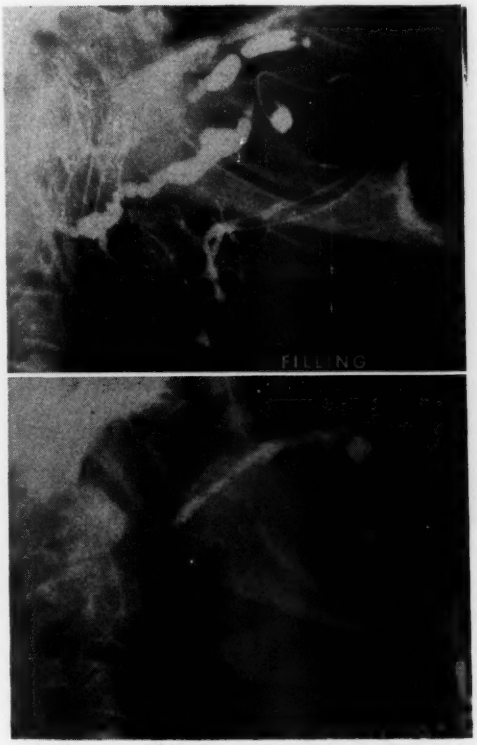

Figure 5.—Sialogram of inflammatory strictures due to ill-fitting dentures. The filling phase of this quadriglandular sialogram demonstrates the markedly dilated and, irregularly segmented ducts (Stenson's ducts). The submaxillary ducts (Wharton's ducts) can be similarly involved, though the degree of dilation is not so pronounced. There is no alteration of the peripheral duct architecture. The post evacuation film indicates that the obstruction at the outlet (orifice) is the principal deterrent to evacuation.

production of multiple inflammatory strictures (Fig. 5). A cordlike Stensen's duct can usually be palpated along its buccomasseteric course. Upon probing the duct, dilated segments alternate with strictures. A gritty sensation imparted to the probe by the hypertrophic and metaplastic duct epithelium may be mistaken for calcareous material.[5]

The diagnostic features of the carbonate apatite salivary calculus [6] affliction as well as stricture formation is main duct obstruction without peripheral duct involvement. This serves to differentiate chronic obstructive sialodochitis from recurrent nonobstructive sialadenitis with sialectasis.

Chronic Non-Obstructive Sialodochiectasis includes a heterogeneous, but clinically related group of diseases which have a similar and characteristic sialographic-histologic pattern known as "sialectasis". These entities include Sjögren's syndrome, recurrent pyogenic parotitis in children and adults and Mikulicz's disease. Both the parotid and submaxillary salivary glands may be affected.

Recurrent tense swelling of the parotid glands is the predominant symptom in this disease group. Although the parotid swelling is usually bilateral, one side may be more severely involved in some cases. Pressure on the gland may cause frank pus or plugs of turbid gelatinous saliva to appear at the orifice of Stensen's duct. Bacteriologic examination reveals the pneumococcus or the streptococcus in pure culture. The patterns of sialectasis are diagnostic and may be identified histologically as well as radiologically and classified into four stages: [2]

1. Punctate sialectasis, in which the earliest findings are characterized by a diffuse punctate dilation of the peripheral ducts which may be less than 1 mm. in size (Fig. 6). With recurrent episodes of parotitis the interlobular ducts are narrowed by periductal lymphocytic infiltration and septal fibrosis. Functioning acinar tissue continues to secrete against a narrowed duct system resulting in retention of saliva in the intralobular ducts which then dilate.[2]

2. Globular sialectasis, in which the dilation of the peripheral ducts increases, but with uniform size and distribution (Fig. 7). It is at this stage that the sialographic findings of terminal duct dilation and lack of intraglandular branching correlate with the scarcity of interlobular ducts located in the septae of the gland and the wide lumens of intralobular ducts in the histologic sections.[2]

3. Cavitary sialectasis, in which there is coalescence of the globules, which now become irregular in size and distribution and decrease in number. These coalesced

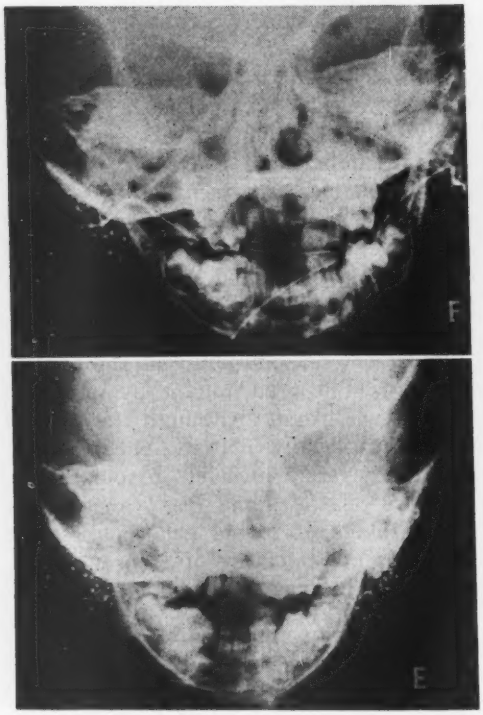

Figure 6.—Sialogram of recurrent pyogenic parotitis in children. The filling phase (top) demonstrates diffuse punctate dilation of the peripheral ducts and a striking decrease in the number of more proximal intraglandular ducts. In the five minutes postevacuation film (bottom) there is retention of contrast material in the dilated terminal ducts.

intralobular ducts continue to dilate and form cysts, resulting in the cavitary appearance noted on sialograms (Fig. 8).

4. Destructive sialectasis, the end stage, in which there is a bizarre pattern which suggests invasion and destruction of the gland parenchyma by neoplasm. Histologic sections support the roentgen findings in that there is no recognizable duct structure, because of lymphoid infiltration and fibrosis of diffuse areas of gland parenchyma. Differentiation from neoplastic disease can be made by demonstration of multiglandular involvement at this phase of sialectasis (Fig. 9).

The management of patients with sialectasis has included treatment with antibiotics, x-ray therapy and steroid hormones. The controversy regarding the

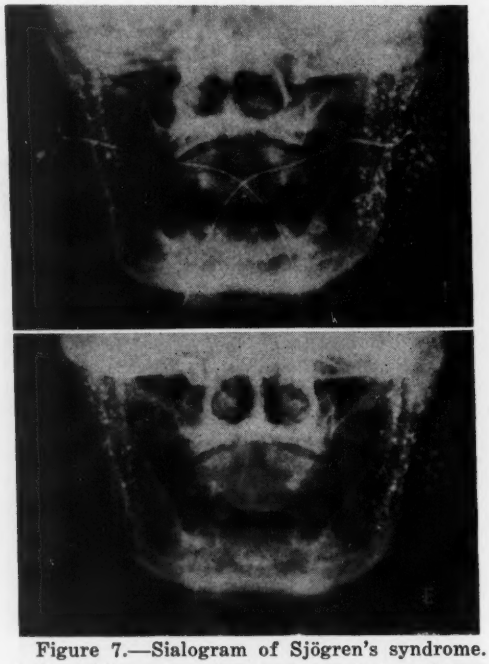

Figure 7.—Sialogram of Sjögren's syndrome. In the filling phase (top) the typical mulberry pattern of globular sialectasis is present. In the half hour postevacuation film (bottom) the scanty salivary flow observed clinically is verified by the unaltered appearance of the dilated peripheral ducts.

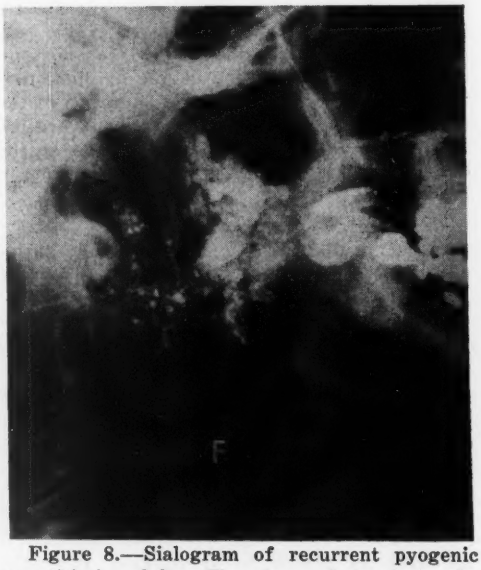

Figure 8.—Sialogram of recurrent pyogenic parotitis in adults. The above photo shows the filling phase. The parotid gland shows irregular cystic cavities replacing the normal duct pattern.

Figure 9.—A sialogram which demonstrates involvement of the submaxillary and parotid glands by nonobstrutcive sialodochiectasis in Sjögren's syndrome. The above photo shows the filling phase. The parotid gland architecture is bizarre and compatible with the destructive stage of sialectasis. The submaxillary sialectasis is of lesser degree.

true nature of the disease entities which comprise the sialectasis group should be resolved within the near future as investigation goes forward into the etiology and pathogenesis.

Other Inflammatory Diseases. Persistent, usually asymptomatic enlargement of the major salivary glands due to nutritional insult is being recognized with increasing frequency. This condition, which appears to involve the parotid glands in particular, is associated with disturbed nutrition [7-9] as seen in obesity; diabetes mellitus with liver disease; [10-11] achalasia and anorexia nervosa; [12] alcoholism with or without cirrhosis,[12-14] as well as inadequate diet due to poverty or custom. The nutritional insult may be continuing or remote. Patients so afflicted may have a watery, yet scanty salivary flow. The volume of pure saliva secreted in ml. per hour may be decreased.[15] Thus the mechanism which resists retrograde infection of salivary gland parenchyma by mouth organisms—i.e. normal salivary flow is impaired and episodes of suppuration may ensue.

Morphologic observations in the literature are few and conflicting. We have found repalcement of secreting gland parenchyma by adipose tissue.[16] The remaining acinar cells may be increased in size; [17] there may be prominence of zymogen granules and eosinophilic reaction of duct epithelium,[16] a finding associated with protein deficiency observed in the laboratory.[18] The histologic finding of fatty infiltration along with decrease in secreting gland parenchyma explains the diminution of total terminal (peripheral) duct mass noted on sialograms which are otherwise normal.

In addition to a review of the dietary history and evaluation for diabetes, alcoholism and liver disease, these patients should be examined by sialography and parotid biopsy so that more can be learned about this clinical entity.

The major salivary glands, particularly the parotids are also subject to involvement by the specific granulomata, although these are relatively uncommon. Boeck's sarcoid, actinomycosis and tuberculosis are the ones with which this author has had personal experience. Specific granulomata involving the parotid lymph nodes such as cat scratch fever and tularemia should not be confused with intrinsic parotid granulomata. In each instance there is usually a primary face or scalp lesion.

Lesions Extrinsic to the Major Salivary Glands

Tumors external to the major salivary glands are more often the cause of regional swelling about the angle and below the ramus of the mandible than are intrinsic salivary gland neoplasms. Inflammatory lymph nodes, metastatic adenopathy and lymphomas are frequently seen as painless masses in the cervicofacial region. Neoplasms derived from the neighboring tissues, any of which may be a potential cause of swelling are problems in differential diagnosis. These include neurofibroma, lipoma, lymphangio-

ma, chemodectoma, branchiogenic cysts and mandibular osteoblastoma. Masseter hypertrophy should also be considered.

Sialography can serve as a diagnostic aid in identifying inflammatory and neoplastic lesions adjacent but extrinsic to the salivary glands. The filling phase shows displacement, compression or distortion of the duct system by the adjacent extrinsic mass but the general architecture is preserved.[19] Evacuation of the contrast material is complete in the emptying phase (Fig. 10). This is the differ-

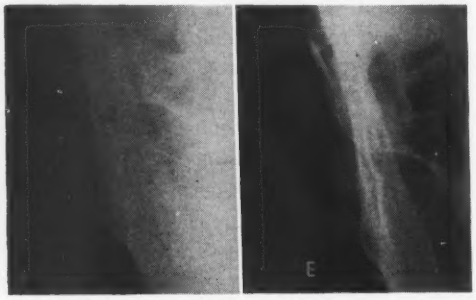

Figure 10.—Sialography in tumors external to the major salivary gland. In the filling phase there is a medial filling defect of the parotid gland. The postevacuation film indicates that the gland has emptied completely and is not involved.

ential point between extrinsic and intrinsic salivary gland neoplasms, since the latter usually show retention of contrast material in post-evacuation films.

Intrinsic Neoplasms

Neoplasms of the major salivary glands are principally confined to the parotid gland. The majority of parotid tumors are epithelial in origin and may be divided into benign and malignant types according to the classification [20] in Table 1.

The presence of a neoplasm in the parotid gland is usually rather obvious although its external manifestations will vary according to the position within the gland which it occupies. Thus a tumor originating in the pharyngeal prolongation of the parotid gland may assume considerable size and even produce bulging into the lateral angle of the pharynx before it causes significant external swelling back

TABLE 1
NEOPLASMS OF THE MAJOR SALIVARY GLANDS

Benign Epithelial Tumors

- I. Mixed tumors
  - 1. Stromal elements predominating
    - a. Cylindromatous type
  - 2. Epithelial elements predominating
    - a. Canalicular type
- II. Mucoepidermoid tumors
- III. Adenomas
  - 1. Serous cell
  - 2. Acidophilic cell
    - a. Papillary lymphoid cystadenoma

Malignant Epithelial Neoplasms

- I. Carcinomas
  - 1. Squamous cell carcinomas
    - a. Cornifying
    - b. Noncornifying
    - c. Lymphoepithelioma
  - 2. Gland-celled carcinomas
    - a. Pseudoadenomatous basal cell carcinoma-cylindromatous, canalicular or medullary, and cystic forms
    - b. Acidophilic gland-celled carcinoma
    - c. Serous cell adenocarcinoma
  - 3. Mucoepidermoid carcinoma
  - 4. Unclassified carcinoma

of the angle of the mandible and protrusion of the lobule of the ear.

Purely clinical differentiation between benign and malignant parotid tumors is usually difficult preoperatively. Although pain is much more common in malignant tumors, the symptom cannot be relied upon for differential diagnosis. The only positive local sign of malignancy preoperatively, is facial paralysis. A benign tumor even of enormous size may produce great stretching and displacement of the facial nerve, but rarely, if ever causes paralysis. In general, it may be said that well encapsulated tumors are benign. The exception to the rule is the pseudoadenomatous basal cell carcinoma or the malignant cylindroma, a highly lethal though slowly growing tumor which may appear to be well encapsulated. Conversely, the nonencapsulated apparently infiltrating tumors are usually malignant but again an exception lies in the benign mucoepidermoid tumors whose surrounding inflammatory reaction gives the gross path-

ological impression of a widely infiltrating malignancy.

Sialography is a useful diagnostic aid in differentiating between intrinsic neoplasms, benign and malignant — in that generally speaking, the benign neoplasm is usually encapsulated or circumscribed whereas the infiltrating neoplasm is usually malignant. Utilization of the filling phase of sialography alone makes it diffi-

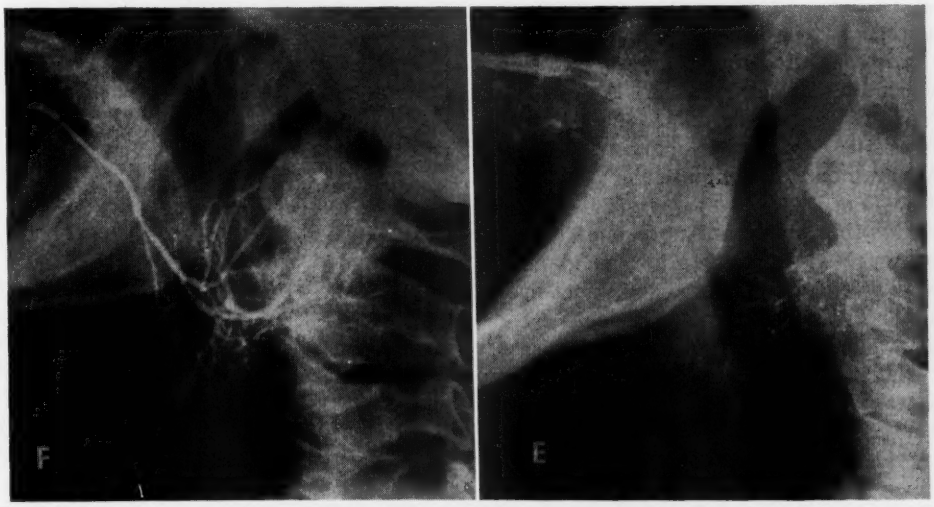

Figure 11.—Sialogram of an intrinsic circumscribed or encapsulated tumor. The filling phase (left) demonstrates a filling defect in the superior pole of the parotid gland with displacement of the gland inferiorly. There is slight dilation of the distal portions of the interlobar ducts.

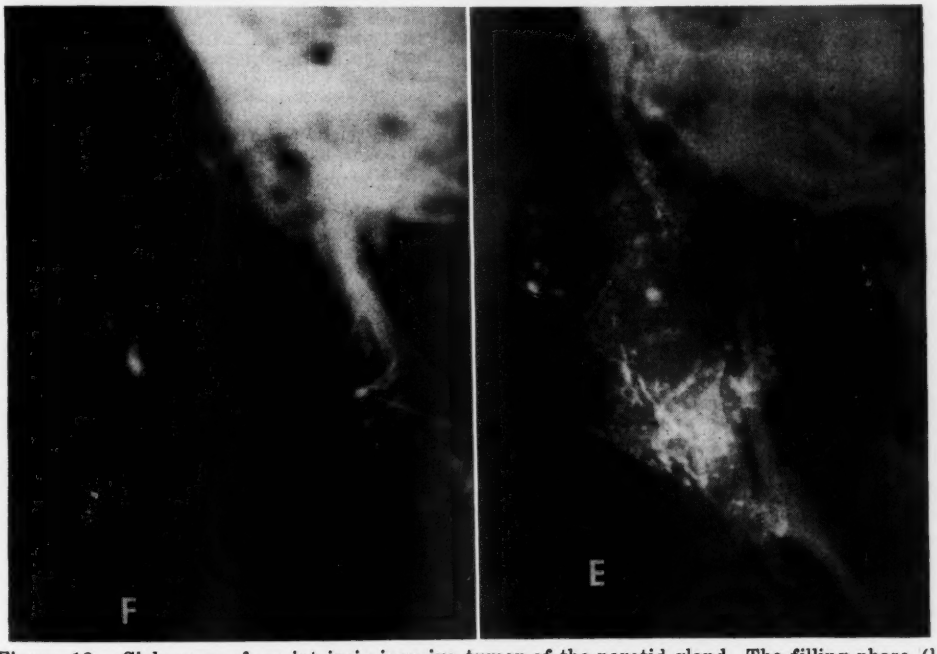

Figure 12.—Sialogram of an intrinsic invasive tumor of the parotid gland. The filling phase (left) demonstrates a large mass in the right parotid gland displacing and altering the duct architecture. Following stimulation (right) the contrast material appears to have permeated the surrounding acinar tissue instead of being evacuated from the gland.

cult to distinguish extrinsic tumors from intrinsic circumscribed or encapsulated neoplasms in which the glandular architecture is also preserved. However, in the postevacuation films, retention of contrast material in distinguishing patterns is noted.[2, 4] The normal gland tissue is compressed between the slowly growing encapsulated or circumscribed neoplasm and the capsule of the gland. This results in patterns which are characterized by some segmental intraglandular duct retention (Fig. 11).

Infiltrating malignant neoplasms of the salivary glands may disrupt the glandular architecture and destroy the duct system. The acinar pattern is replaced by a bizarre puddling and pooling of contrast material (Fig. 12). The postevacuation phase shows virtually complete retention of the contrast material.[2, 4] It should be reiterated that these bizarre puddling and pooling patterns are indistinguishable from the radiographic picture of destructive sialectasis. The differential point lies in the fact that the other major salivary glands, exclusive of the one involved by a tumor, show the normal sialographic pattern. Certainly no attempt should be made to diagnose the histologic type of salivary gland neoplasm by sialography.

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Acute Chyloperitoneum; A Case Report*

• The authors give a review of the literature and report the fourteenth case of acute idiopathic chyloperitoneum.

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A SURVEY of the literature discloses 38 reported cases of acute chyloperitoneum of which 13 were thought to be idiopathic.[1-9, 11, 13] A recent case of idiopathic chyloperitoneum at Charity Hospital is added.

Although relatively infrequent, a number of cases of chylous ascites, some associated with chylothorax, have been reported since 1694 when Morton gave the classical description of this condition.[4] Extremely rare are those cases in which the presence of effused chyle in the peritoneal cavity is associated with acute onset of symptoms. Renner, as cited by Hoffman,[4] published in 1910 the earliest report of such a case. In 1954 Hoffman [4] presented a collection of 23 similar cases and added one of his own. He designated the condition as chyle peritonitis. While obstruction or trauma were the underlying factors in many of these cases, no etiology was found in 10 of the 24. Since Hoffman's report, 3 additional cases of acute idiopathic chyloperitoneum have been described.[1, 6, 9]

Nix et al [12] in 1957 reviewed 302 cases of chylothorax and chylous ascites, and analyzed 11 cases from Charity Hospital at New Orleans. Five of these involved the abdomen only, 4 involved the abdomen and the chest, and 2 involved the chest only. Since this report, 3 additional cases involving the abdomen and evidencing chronic symptoms were seen at this hospital. The first of these 3 cases was a 2 year old white female who had chylous ascites at the age of 2 weeks and was treated with multiple paracenteses. Follow-up of more than one year has shown no recurrence. The second patient, a 51 year old colored male with chylous ascites, evidently from tuberculosis, has been placed on antituberculosis therapy. The third case was a 34 year old colored male who was found to have 300 to 400 cc. of milky fluid in a hernial sac at the time of an inguinal hernioplasty. Two months following operation he died with metastases from a reticulum cell sarcoma. While there have been 14 cases of chronic chylous ascites at Charity Hospital, the subject of this report is the only patient found to have free chyle in the peritoneal cavity with acute onset of symptoms.

Case Report

H.N., a 47 year old colored male, was seen in the Admitting Room of Charity Hospital on October 20, 1959, complaining of pain in the stomach. He had been well until two days prior to admission when he experienced intermittent cramping epigastric pain associated with a desire to expel flatus. Later in the day the pain became constant and was described as a "dull tight feeling across the upper abdomen". By the morning of the second day the pain was much worse and radiated into the right lower quadrant. At this time soreness appeared at the base of the neck on the left. There was no history of any serious illness, and the patient had never been operated upon. He had always worked as a laborer.

Physical examination revealed a blood pressure of 150 systolic, 80 diastolic, pulse 84, respiratory rate 20, and temperature 99°F. He appeared to be in no acute distress but complained of abdominal pain. There was moderate tenderness over the left lower neck with a few palpable lymph nodes. The entire abdomen was

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tender, especially in the right lower quadrant and the epigastrium. There was referred rebound tenderness to the right lower quadrant. No palpable masses were present and peristaltic sounds were normal. There was tenderness in both costovertebral angles. A large hydrocele was present on the left.

Roentgenograms of the chest and abdomen were not revealing.

The hematocrit was 46%, white blood cell count was 6,700 with 70% polymorphonuclear leukocyte, 6% bands and 24% lymphocytes. Urinalysis was negative. Amylase was 181 units/100 ml., fasting sugar 84 mg/100 ml., blood urea nitrogen 14 mg./100 ml. Electrocardiogram was normal.

Admitting diagnosis was ruptured peptic ulcer versus ruptured appendix.

Within a few hours the pain and tenderness had become more localized in the right lower quadrant, and a presumptive diagnosis of acute appendicitis was made.

At operation 200 cc. of milky fluid were found in the peritoneal cavity and there were several dilated loops of small intestine. In addition, white patches were present within the small bowel mesentery and in the wall of the jejunum. No other abnormalities were noted. Appendectomy was performed, and the abdomen closed without drainage.

Smear of the abdominal fluid showed a few mesothelial cells and lymphocytes but no bacteria. A biopsy was taken of the lymph nodes at the base of the neck on the left and reported as nonspecific chronic lymphadenitis. Abdominal paracentesis one week after operation yielded 2 cc. of clear peritoneal fluid. The patient was discharged on a low fat diet to be followed in the outpatient clinic.

Discussion

There have been various views as to the action of chyle. Some have thought it to be bacteriostatic, and others have described chyle as an irritant in the peritoneal cavity.[4] Meade [10] mentioned the local effect of chyle on the pleura with loss of elasticity and deposition of a layer of exudate on the pleural surface. Freeman and Johnson [3] described the hemolytic action of chyle but gave evidence to show that fasting animals contained an insufficient amount of fatty acid to produce hemolysis. In the majority of reported cases of acute chyloperitoneum, symptoms have followed shortly after a heavy meal. This may be coincidental, yet it does suggest that symptoms are caused by lymph containing fatty acids.

The patient presented showed several features thought to be characteristic of chyle peritonitis. There was acute onset of pain following a large meal. When first examined, the patient was thought to have had a perforated ulcer or acute appendicitis. There was free chyle in the peritoneal cavity with subserosal plaques and edematous mesentery of the small bowel. The postoperative course was benign, and the patient received no specific therapy. The finding of lymphadenopathy and chronic lymphadenitis of the cervical nodes is of interest, but merely suggests that this could have been a contributing factor.

Treatment of chronic chylous ascites has usually consisted of repeated paracenteses and specific therapy directed toward the underlying condition. In acute chyloperitoneum, where the diagnosis is made at the time of operation, simple evacuation of the chyle and closure without drainage has been adequate treatment. This method has been successful in all but one reported case.[4] Postoperatively, the majority of patients have been placed on low fat diets.

Whether acute chyloperitoneum is an entity is not known. The association with such things as intestinal obstruction, trauma, and congenital lymphatic anomalies, would suggest that it is simply one manifestation of several different underlying pathological processes. The explanation for the idiopathic variety obviously may be that the etiology has remained obscure. That it is related to heavy ingestion of fatty foods in some way is possible but not proven. The cause of acute symptoms is not definitely known.

Summary

1. The fourteenth case of acute idiopathic chyloperitoneum is presented.

2. The diagnosis of this condition is made at the time of operation, the typical findings being free chyle in the peritoneal cavity, subserosal plaques in the small bowel, and a thickened small bowel mesentery.

3. While the role played by the chyle in the production of the acute symptoms is unknown, it has been shown that chyle has a hemolytic action probably due to free fatty acids.

4. Treatment consists of evacuation of the chyle. Drainage is probably not necessary.

5. Prognosis in this condition has been good.

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Women as Physicians

There is nothing like logic of facts. To it all theories must be brought and tested. It is vain to attempt to escape its verdict.

The question whether women are calculated to be successful in the profession has been debated vigorously. Now is the time we can ask, **have they been** successful? The record is before us and open for inspection.

The Valedictory Address of Dr. Rachel L. Bodley, Dean of the Woman's Medical College of Philadelphia, placed this matter before her hearers. Her theme was a statement of the work accomplished by their Alma Mater in the thirty years of the existence of the College, as exemplified by the professional careers of her 276 daughters sent out as medical graduates during that time. To enable herself to speak with precision, Dr. Bodley sent to each of the 244 surviving graduates a circular containing eight questions in the practice of their life work. * * * its professional character; its pecuniary rewards; its social status; its teaching work; its membership in medical societies, and finally that important question in sociology touching the influence of the study and practice of medicine upon woman's relation as wife and mother.

* * *

The fifth question relates to the monetary value of the medical practice per year, and is answered by 76 ladies.

24 as much as $1,000, and less than $ 2,000
20 as much as $2,000, and less than $ 3,000
10 as much as $3,000, and less than $ 4,000
5 as much as $4,000, and less than $ 5,000
3 as much as $5,000, and less than $15,000
4 report sums varying from $15,000 to $20,000 per year.
10 report less than $1000 per year.

New Orleans M. & S. J. 8:222 (Sept.) 1881

Cat-Scratch Disease--Another Cause Of Parinaud's Oculoglandular Syndrome

• The author describes three cases of this disease, which is becoming more generally recognized.

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SINCE 1889, when Parinaud first described the oculoglandular syndrome, it has been steadily broken down into its component diseases. The newest etiological agent to be described is the virus of cat-scratch fever. The purpose of this paper is to present two well documented cases of the syndrome complete with antigenic and pathological studies. No attempt to review the literature or describe the disease will be made, as reference to the bibliography will suffice.

Case Presentations

Case No. 1.—D. P., a white male, aged 15, was seen complaining of a mass under the right upper lid of one and a half weeks' duration. Fever (undetermined) had been present the first three days, and a right preauricular node appeared one week previously. Five weeks before, patient had had contact with a tuberculous patient. He had played frequently with pet cats at home. None were ill, but two months previously a pet white mouse became ill, showing blindness, deafness and enlarged neck and groin glands. It was killed by the Society for the Prevention of Cruelty to Animals. The patient's brother has Pott's disease.

Examination revealed a mushroom shaped mass 4 by 4 by 1 mm. under the right upper lid, attached laterally near the upper border of the tarsal plate by a base 0.5 cm. in diameter. This mass, composed of rather solid homogenous reddish tissue, was not ulcerated. A mild mucopurulent conjunctival discharge was present, as well as local conjunctival inflammation. The enlarged preauricular node (1 by 1 by 1.5 cm.) was not tender or fluctuant.

During hospitalization the following tests were done:

X-ray chest—negative.

Sed. rate 24 mm., Hb. 12.5 (79.4 gm.). RBC 4,100,000, Color Index (bld. .96). WBC 5,350 (Neut. 55, Eosin, 3, Lymphs. 39, Monos. 3).

Urine—negative.

Agglutinations: Typhosus 0 & H, Para A and B, Brucella, Proteus X-19, all negative 1:40.

Glucose 97. NPN 27.

Wassermann and VDRL—negative.

Conjunctival smear for AFB and fungi—negative. Conjunctival smear for fusiform bacilli—negative, with crystal violet. Conjunctival smear for Spirochetes (ink preparation)—negative. Routine conjunctival smear and culture — negative twice.

Agglutinations for tularemia—negative 1/20.

Dark Field—negative for spirochetes from conjunctival lesion.

Conjunctival smear for eosinophiles—negative.

Scrapings from eye lesion and conjunctiva (Wet mount no fungi) and fungus culture—negative.

A complete general physical examination was entirely normal, except for ophthalmological findings, enlarged preauricular node, enlarged axillary nodes and shotty posterior cervical adenopathy.

Only one fever spike to 101.6° was noted in 2 five day hospital stays.

Differential Diagnoses

It may be interesting to note the differential diagnoses considered by three physicians involved:

A. Internist.
   1. Non-specific conjunctivitis with adenopathy.
   2. Boeck's Sarcoid.
   3. Lymphoma—leukemia.
   4. Tuberculosis.
   5. Cat-scratch Disease.

B. Consulting Ophthalmologist.
   1. Tularemia.
   2. Vernal Catarrh.
   3. Cat-scratch Disease.
   4. Leptothrix conjunctivitis.

C. Ophthalmologist.
   1. Cat-scratch Disease.
   2. Tularemia.

3. Tuberculosis.
4. Sporotrix—Leptothrix.
5. Agricultural conjunctivitis.
6. Beal's conjunctivitis.
7. Conjunctivitis due to *Micrococcus polymorphicus necroticans* of Pascheff.

Biopsy

The entire polypoid mass was shaved from the conjunctiva as a biopsy. The following report was received:

Microscopic Description:

"Sections show a polypoid piece of tissue covered by benign squamous epithelium. It consists of many newformed blood vessels with an inflammatory infiltrate as seen in pyogenic granuloma. Schiff stains are negative for fungi."

Diagnosis: Pyogenic granuloma.

Cat-scratch antigen * was obtained and skin test gave a positive reaction with a hard red central papule and a zone of erythema extending out 1½ inches. The preauricular node was almost normal size at that time, and the conjunctival lesion healed.

Case No. 2.—P.L., white female, aged 12, complained of a right preauricular node having formed ten days previously. Two days later submaxillary nodes on the right began to swell and eight days later a conjunctival ulcerative lesion was discovered. There had been no fever. As pets, the patient had a parakeet, 2 dogs, a cat, and a snake.

The patient was hospitalized and a general examination was noncontributory, except for the enlarged nodes and conjunctival lesion. No fever was noted. The following laboratory tests were performed:

X-ray chest—negative.

Cat-scratch test strongly positive at forty-eight hours.*

Tuberculin test 1:10,000—negative.

Histoplasmin skin test—negative.

Biopsy showed the conjunctival lesion to be diffusely infiltrated with lymphocytes, plasma cells and neutrophiles. No tubercles were seen. Sections of cervical node showed a severe chronic granulomatous lymphadenitis with large collections of neutrophiles surrounded by a thick zone of macrophages, epitheloid cells, and plasma cells. Schiff and acid fast stains—negative for AFB and fungi. Diagnosis: Chronic granulomatous conjunctivitis with cervical adenitis.

Culture of conjunctival scrapings — negative for AFB four times.

Routine S & C—negative OD.

Agglutinations — Brucella — negative (1/40). Heterophile—negative (1/7). Tularemia—negative (1/20).

Kolmer and Kahn negative.

Blood Glucose, 130. NPN, 28.

Hb. 13.0 (83.2%), RBC 4.3 million. Color index .96.

WBC 7,950, Neutrophils 60, Eosinophils 2, Lymphocytes 36, Monocytes 2.

Urine negative.

Conjunctival culture O.V.—negative twice.

Lymph node S & C—negative twice.

Lymph node anaerobic culture—negative.

Lymph node smear—negative AFB.

Case No. 3.—Simultaneously the brother of the patient developed right axillary adenopathy with subsequent suppuration. Test with cat scratch antigen was positive. There were no ocular findings.

Conclusions

Another etiological agent causing Parinaud's oculoglandular syndrome is presented along with 3 case reports. Cognizance of this agent will result in more accurate diagnosis of this self-limited disease.

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* Antigen supplied by Drs. W. B. Daniels and F. G. MacMurray.

The Journal of the Louisiana State Medical Society

Established 1844

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Subscription Terms: $4.00 per year in advance, postage paid, for the United States; $5.00 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

The Journal does not hold itself responsible for statements made by any contributor.

Editorial

Benjamin Franklin On Politics And Economics

Two hundred years ago, the efforts of government to neutralize the effects of economic laws by legal actions brought forth the displeasure and censure of Benjamin Franklin. Of all the men in the remarkable group among whom he worked he "embodies more and more characteristic qualities which we have agreed to call American than does any other public figure."

The following excerpts from a Benjamin Franklin essay "On the Price of Corn and Management of the Poor" are as pertinent now as they were when written.

"To the Public"

"I am one of that class of people, that feeds you all, and at present is abused by you all; in short I am a farmer.

"By your newspapers we are told, that God has sent a very short harvest to some other countries of Europe. I thought this might be in favour of Old England; and that now we should get a good price for our grain, which would bring millions among us, and make us flow in money; that to be sure is scarce enough.

"But the wisdom of government forbade the exportation.

" 'No'; says my lords the mob, 'you shan't have that. Bring your corn to market if you dare; we'll sell it for you for less money, or take it for nothing.'

"Being thus attacked by both ends *of the constitution*, the head and tail of *government*, what am I to do?

"Must I keep my corn in the barn, to feed and increase the breed of rats? Be it so; they cannot be less thankful than those I have been used to feed."

"Now, if it be a good principle, that that the exportation of a commodity is to be restrained, that so our people at home may have it the cheaper, stick to that principle, and go through-stitch with it. Prohibit the exportation of your cloth, your leather, and shoes, your iron ware, and your manufactures of all sorts, to make them cheaper at home. And cheap enough they will be, I will warrant you; till people leave off making them."

"You say, poor labourers cannot afford to buy bread at a high price, unless they

had higher wages. Possibly. But how shall we farmers be able to afford our labourers higher wages, if you will not allow us to get, when we might have it, a higher price for our corn?"

"But it seems, we farmers must take so much less, that the poor may have it so much cheaper.

"This operates, then, as a tax for the maintenance of the poor. A very good thing you will say. But I ask, Why, a partial tax? Why laid on us farmers only? If it be a good thing, pray, Messieurs the Public, take your share of it, by indemnifying us a little out of your public treasury. In doing a good thing, there is both honour and pleasure; you are welcome to your share of both.

"For my own part, I am not so well satisfied of the goodness of this thing. I am for doing good to the poor, but I differ in opinion about the means. I think the best way of doing good to the poor, is, not making them easy *in* poverty, but leading or driving them *out* of it. In my youth, I travelled much, and I observed in different countries, that the more public provisions were made for the poor, the less they provided for themselves, and of course became poorer. And, on the contrary, the less was done for them, the more they did for themselves, and became richer. There is no country in the world where so many provisions are established for them; so many hospitals to receive them when they are sick or lame, founded and maintained by voluntary charities; so many almshouses for the aged of both sexes, together with a solemn general law made by the rich to subject their estates to a heavy tax for the support of the poor. Under all these obligations, are our poor modest, humble, and thankful? And do they use their best endeavors to maintain themselves, and lighten our shoulders of this burthen? On the contrary, I affirm, that there is no country in the world in which the poor are more idle, dissolute, drunken, and insolent. The day you passed that act, you took away from before their eyes the greatest of all inducements to industry, frugality, and sobriety, by giving them a dependence on somewhat else than a careful accumulation during youth and health, for support in age or sickness.

"In short, you offered a premium for the encouragement of idleness, and you should not now wonder, that it has had its effect in the increase of poverty. Repeal that law, and you will soon see a change in their manners. *Saint Monday* and *Saint Tuesday will soon cease to be holidays. Six days shalt thou labour,* though one of the old commandments long treated as out of date, will again be looked upon as a respectable precept; industry will increase, and with it plenty among the lower people; their circumstances will mend, and more will be done for their happiness by inuring them to provide for themselves, than could be done by dividing all your estates among them.

"Excuse me, Messieurs the Public, if, upon this *interesting* subject, I put you to the trouble of reading a little of *my* nonsense. I am sure I have lately read a great deal of *yours,* and therefore from you (at least from those of you who are writers) I deserve a little indulgence.

"I am yours, &c ARATOR"

The Autobiography of Benjamin Franklin & Selections from His Other Writings, The Modern Library, Random House, Inc. 1950, New York.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

FIVE REASONS WHY MEDICINE IS OPPOSED TO FORAND-TYPE LEGISLATION

1. It is totally unnecessary. The Kerr-Mills Law will do the job.

2. It would mean poorer, not better health care for the aged, with government employees telling doctors what drugs and treatment they could provide their patients; telling hospitals how to operate; telling nursing homes what they could and could not do.

3. It would lead to the decline, if not the end, of private health insurance, which has made such great strides in recent years.

4. It would mushroom into compulsory national health insurance for every American, as time went by. What would start out as socialized medicine for the aged would eventually become socialized medicine for every man, woman and child in the country.

5. It would be staggeringly expensive. Social Security taxes are already scheduled to reach nine per cent of payroll by 1969. This would increase them further. It is well to remember that such bills as these are irreversible in nature. Once started—like an avalanche—they are hard to stop. The tendency is to expand them—never to contract them. As time went on, Congress would face continual pressure for more extended coverage and more elaborate benefits. Costs would rise proportionately.

FIVE REASONS WHY THE KERR-MILLS LAW MERITS PUBLIC SUPPORT

1. The Kerr-Mills Law provides legislation under which it is possible for the first time to help **every** aged American who needs help to pay for health care. Its benefits, based on need and local determination, are much broader than under proposed Forand-type legislation.

2. The program is voluntary, not compulsory. It will supplement, not supplant, individual voluntary health insurance or prepayment.

3. It will be administered on a local basis. The Kerr-Mills Law leaves matters up to the States by giving them the right to set up their own programs in their own way, on the logical theory that each State knows its own particular problem better than the Federal Government does.

4. The new law is economical. Local administration, local choice of scope of benefits and method of payment, local definition and determination of eligibility, minimum Federal control — all these mean that our tax dollars will be spent to best advantage. The use of this approach not only guarantees that we will be helping those who need help, but that we will not be wasting tax dollars on help for those who are perfectly willing and able to take care of their own health costs. And there are millions of these people.

5. The quality of medical care under such a plan will be infinitely superior to that possible under a Forand-type approach. Under the new law, any type of care can be provided if the State determines the need for it. Under the Forand Bill, only hospitalization, nursing home care, and some surgical care would be authorized. Further, the fact that Federal controls are held to a minimum allows adequate supervision of the program by physicians, and no restraints upon the doctor in the exercise of his medical judgment. The medical profession's basic policy has always been to insure that the needy receive the same high quality of medical care as anyone else in the community. Under the Kerr-Mills Law, this can and will be accomplished, whereas it could not be under the restrictions imposed by Forand-type legislation.

FOR THE INFORMATION OF OUR MEMBERS

Those who favor the unsound and dangerous Forand Bill are now exerting a concerted effort to pass, with President Kennedy's support, some Forand type legislation controlled and operated under Social Security regulations with Social Security taxes. Under this plan an agency of the Federal Government operating out of Washington, D. C., would cover millions of people who neither need nor want government help under a system of compulsory national health insurance.

This approach was considered at the last Congress and soundly rejected. However, its backers are not willing to give the Kerr-Mills Bill duly enacted by the last Congress a chance to prove its effectiveness in taking care of our oldsters who really need and want help for their medical care.

Representative Aime Forand (D-R.I.), father of the Social Security medical plan bearing his name, is quoted in the January 12 Chicago Daily News as saying, "If we can only break through and get our foot inside the door, then we can expand the program after that."

The Forand type legislation was defeated in the last Congress by the vigorous campaign waged by the medical profession and its loyal allies. If it is still our wish to keep this country free from socialized medicine, we must again wage a most vigorous and united campaign against this vicious legislation, which if enacted into law will eventually and completely socialize not only the medical profession, but all of our professions.

The Forand type legislation implemented and financed under the Social Security system would provide only limited health care of inferior character to only a limited number of our old people. This service would not be available to millions of our older citizens who need medical services.

It is the duty of every physician, if he hopes to continue to practice medicine under a free enterprise system, to write his Congressman and Senator stating his or her opposition to any Forand type legislation including medical care under the Social Security system. Give reasons for your opposition: It would mean federally controlled medicine of poor quality. There would be a marked reduction of private health insurance which has done so much for the protection of our citizens during the past few years. Increasing numbers of our people have availed themselves of this source of protection. The cost of implementation will be almost prohibitive. It is estimated that by 1969 Social Security taxes will reach 9% of the payroll, and will continue to increase as more expanded benefits are offered. Costs would continue to rise as Congress is pressured into extended coverage and more elaborate benefits.

Such legislation is wholly unnecessary and will not cover, as stated above, millions of our citizens who really need medical service and great numbers of our citizens who do not need it and do not want it will be forced to take it because it is a compulsory federal program.

The Kerr-Mills law already prepared by Congress is a voluntary service and will do the job better and cover more patients who need this service than the governmental compulsory and socialistic Forand type legislation which the new administration in Washington is trying to force on our old citizens.

GOOD NEWS IS SO SELDOM RECEIVED

Our attorneys have just notified us that the Court of Appeal, State of Louisiana, has refused to grant a rehearing to the Chiropractors in their suit to declare our Medical Practice Act unconstitutional.

Their next move, we presume, will be to appeal the case to the State Supreme Court. Since this Court has, on other occasions, held this Act to be constitutional, we are hopeful that this litigation will be closed at an early date, and that the Board of Medical Examiners will be freed from the injunction which has prohibited further prosecution, and will be free to again prosecute these offenders in the State Courts.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Ascension	Third Tuesday of every month	
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Lafayette	Second Tuesday of every month	Lafayette
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SOUTHERN REGIONAL MEETING OF THE AMERICAN COLLEGE OF GASTROENTEROLGY

The Southern Regional Meeting of the American College of Gastroenterology will be held in Houston, Texas on Sunday, 19 March 1961. The sessions, which will be held in the Jesse H. Jones Library Building of the Texas Medical Center, will commence at 9:00 a.m.

Participating in the program will be among others, W. C. Arnold, M.D., Robert Nilson, M.D., George Morriss, M.D., and Bela Halpert, M.D., F.A.C.G., Houston, Texas; H. J. Robert, M.D., Palm Beach, Florida; Henry Laurens, M.D., A. C. Broders, Jr., M.D., Temple, Texas. The Houston Gastroenterological Society will be the host for this meeting.

The program is under the Chairmanship of Ralph D. Eichhorn, M.D., F.A.C.G., Houston, Texas and H. B. Eisenstadt, M.D., F.A.C.G., Port Arthur, Teaxs, Governor of the College for Texas.

The Southern region consists of the states of Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas and Virginia.

Members of the medical profession are cordially invited to attend. A copy of the program may be obtained from the Secretary, American College of Gastroenterology, 33 West 60th Street, New York 23, N. Y.

CHEST PHYSICIANS ESTABLISH FUND TO ASSIST CUBAN MEMBERS

At the meeting of the Board of Regents of the American College of Chest Physicians held in Washington, D.C. on November 28, 1960, a resolution was adopted to establish a relief fund for Cuban members of the College who have been exiled temporarily from their country. The Board of Regents voted to contribute $5,000 to launch the fund and contributions are being solicited from College members and others who are interested. The Cuban Chapter of the College was founded in 1940 and now has 74 members.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination (Part II), oral and clinical for all candidates, will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from April 8 through 15, 1961. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

Candidates who participated in the Part I Examination will be notified of their eligibility for the Part II Examinations as soon as possible.

Current Bulletins of the American Board of Obstetrics and Gynecology, outlining the requirements for application, may be obtained by writing to Robert L. Faulkner, M.D., Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

WESTERN REGIONAL MEETING OF THE AMERICAN COLLEGE OF GASTROENTEROLOGY

The Western Regional Meeting of the American College of Gastroenterology will be held in San Francisco, Calif. on Sunday March 5, 1961. The sessions, which will be held at the Fairmont Hotel, will commence at 9:00 a.m.

Participating in the program will be Dwight L. Wilber, M.D., Kahn Uyeyama, M.D., Hugo C. Moeller, M.D., Warren C. Breidenbach, M.D., all of San Francisco, Calif.; Raymond M. Kivel, M.D., Palo Alto, Calif.; Donald C. Collins, M.D., F.A.C.G., Hollywood, Calif.; and Roger C. Simpson, M.D., San Francisco, Calif.

The program is under the Chairmanship of Samuel W. Yabroff, M.D., F.A.C.G., Oakland,

Calif., Governor of the College for Northern California.

The Western region consists of the states of Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington and Wyoming.

Members of the medical profession are cordially invited to attend. A copy of the program may be obtained from the Secretary, American College of Gastroenterology, 33 West 60th Street, New York 23, N. Y.

ANNUAL MEETING, LOUISIANA CHAPTER
American College of Chest Physicians

The Louisiana Chapter of the American College of Chest Physicians will sponsor a series of case presentations at its annual meeting at the Veterans Administration Hospital, New Orleans, March 10. Cases will be presented by the following physicians: Page Acree, Baton Rouge; Oscar Alvarez, Lafayette; William Fey, Lake Charles; Walter McCook, Shreveport; and Zoltan Mann, Alexandria. The following New Orleans physicians will serve as panel consultants for the case presentations: Oscar Blitz, Howard A. Buechner, Charles Dunlap, Leo Horan, Sydney Jacobs, Charles Nice, Robert Schramel, John H. Seabury, Lawrence H. Strug, Morton Ziskind, and Herman J. Moersch, Chicago, Illinois.

There is no registration fee and all physicians are invited to attend the meeting.

2 NEW ADVISORY MEMBERS APPOINTED AT BANK OF LOUISIANA

The appointment of two businesses and professional leaders to the advisory committee to the Bank of Louisiana in New Orleans is announced Wednesday, February 1, 1961 by James F. Quaid, president.

Leonard J. Elmer, president Elmer Candy Co., Inc. and Dr. John S. Salatich, both of New Orleans, are the new members of the advisory committee.

Elmer is a member of the National Confectioners Association and has been in the candy business practically all his life. His Elmer's candies are one of the better known national brands. He is a past officer and board member of the New Orleans Athletic Club, and has always been actively engaged in other business, civic and social organizaitons.

Dr. Salatich is in the private practice of internal medicine and is chief admitting physician of Charity Hospital of Louisiana in New Orleans. He received his bachelor of science degree from Loyola University of New Orleans in 1946, and his doctor of medicine from Louisiana State University in 1950. Dr. Salatich is a member of the American Medical Association, Louisiana State Medical Society, Orleans Parish Medical Society, and the American College of Physicians.

SOUTHWESTERN SOCIETY OF NUCLEAR MEDICINE

This Society will hold its Sixth Annual Meeting in Oklahoma City, April 8-9, 1961, at the Ramada Inn, under the presidency of Dr. Samuel B. Nadler of New Orleans.

Inquiries concerning this meeting should be addressed to Dr. Peter E. Russo, Program Chairman, 1200 North Walker, Oklahoma City. Doctors and individuals interested in this field are cordially invited to attend. The annual banquet will be held Saturday evening, April 8.

ANNOUNCEMENT

The SCOTT AND WHITE CLINIC announces its NINTH ANNUAL POSTGRADUATE CONFERENCE in **Medicine and Surgery** to be held in Temple, Texas on March 5, 6, 7, 1961. Practical aspects of medicine and surgery will be presented through clinics and open discussions:

CREDIT through the University of Texas Postgraduate School of Medicine to the Academy of General Practice.....18 hours.

Registration Fee — $25.00.

Wives are cordially invited.

Please write for further information.

BOOK REVIEWS

Rudolph Matas — A Biography of One of the Great Pioneers in Surgery; by Isidore Cohn, M. D., with Hermann B. Deutsch. Garden City, New York, Doubleday & Company, Inc., 1960. pp. 431; illus. 24. Price $5.95.

The timing of this biography is noteworthy, in marking the birth centennial of its distinguished subject whose life spanned ninety-seven years of the century. Rudolph Matas died in 1957. But the challenge and inspiration of his life will survive through generations, thanks to the devoted work of Dr. Isidore Cohn and his able collaborator, Mr. Hermann B. Deutsch.

Fifty-four years of close association with Dr. Matas in professional relations (first as a student), and in intimate friendship as well, eminently qualify Dr. Cohn for undertaking the biography. In addition to his personal observations of Dr. Matas, Dr. Cohn has had at his disposal a wealth of revealing source materials,

among them diaries and correspondence entrusted to him by Dr. Matas.

In the preface, Dr. Cohn states: "Years ago, when I first suggested that I be allowed to be his biographer, he demurred because, as he explained it, 'you would be too partial.'" The later placing of confidence in the prospective biographer was fully justified; I say this with knowledge of Dr. Matas gained by my own contacts with him through thirty-seven years. The biography is not partial, for it is not distorted by Dr. Cohn's feeling of affection and his admiration of this teacher, colleague, and friend. Weaknesses are not glossed over and strengths are not magnified; the account is impressively objective. The five-page appendix may be mentioned as an illustration of restraint. These pages are devoted to a simple listing of the numerous recognitions accorded to Dr. Matas in the form of degrees, honorary awards, foreign decorations, and official positions in medical societies. Each entry is only the symbol of a chapter that might have been written.

The volume presents an even, well-rounded life story that gives insight into the facets of Dr. Matas that had been unknown even by many who were well acquainted with him. It presents him not alone as a man of medicine but as boy, youth, and man in his individual pattern of human existence. The writing is vivid and compelling in interest. As we read, Dr. Matas seems to live again. We hear him exclaiming "Confusion" in some trying situation and muttering "My, my, my" while deliberating on it; we hear him again in prolonged discussion of a program paper, often contributing more to the subject than did the original speaker. We see him again, an imposing figure, wholly engrossed in attentive listening, or in speaking, or in doing —always oblivious to the passing of time.

Rudolph Matas was a Louisianian by birth and by virtual life-time residence. He was born, in 1860, on a plantation near New Orleans where his father Narciso, served as physician. His parents, both Spaniards, had left their home country for Louisiana shortly after their marriage in 1856. Narciso Matas had an urge to leave Gerona because of its associations with his dead mother, and his choice of destination was prompted by family ties in New Orleans. Narciso Matas, then not yet twenty years of age, had an adventurous spirit; he welcomed new opportunities wherever they might lead him. He retained that spirit throughout his life. His first venture in New Orleans was the operation of a pharmacy, but within a year he enrolled in the New Orleans College of Medicine, wherein he earned two degrees: the Doctor of Pharmacy in 1858 and the Doctor of Medicine in 1859. Dr. Narciso Matas was a man of many enterprises, including activities in business as well as in the profession. His preoccupations left scant time for attention to the little son Rudolph, who was four years old when he first had really intimate association with the father. At this time Dr. Narciso Matas, with his family, journeyed to Paris for special studies in ophthalmology; here the boy was in a medical environment. The father's fellow students often gathered in the apartment for "shop talk" and for practice in ocular surgery, using eyes from the abbatoir. Rudolph, to the pride of his father, learned some of the anatomical features of the eye. One can only conjecture as to whether these early experiences, including the new paternal understanding, may have influenced the channeling of his eventual choice of career. At any rate, this period is worthy of note because it showed emergence of some of the qualities of his manhood, perceptiveness and exceptional memory.

The four-year-old Rudolph was precocious, and as teen-ager and young man his accomplishments seemed always advanced for his age. His elementary and secondary schooling was interrupted and migratory. His education at the Medical Department of the University of Louisiana (later the School of Medicine, Tulane University) began when he was seventeen and he became Doctor of Medicine before reaching the age of twenty. The early schooling and the professional schooling as well, the latter characteristic of that time, would not have equipped any ordinary person for what he was destined to accomplish.

Rudolph Matas was no ordinary person. His endowments were exceptional, among them great physical vigor, to which he often referred as a cherished inheritance; outstanding intellectual vigor; perceptiveness; phenomenal memory; thoroughness and tenacity of effort on the task at hand. His capital in assets such as these yielded a maximum return because he abhorred time-wasting; he curtailed hours of sleep and made every waking hour productive.

At the age of seven, in an epidemic year when 3107 residents of New Orleans died of yellow fever, Rudolph Matas contracted the dread disease. The immunity thus acquired not only protected him during subsequent New Orleans epidemics but was of particular value when, still a medical student, he spent three months in Havana as a member of the Yellow Fever Commission that sought to discover how the infection is transmitted. The Commission did not solve this problem but it laid groundwork, in large part through Matas' energetic investigations, that served a bit later to substantiate Dr. Carlos J. Finlay's demonstration of the role of the mosquito in passing the agent of disease from an infected person to another. Dr. Finlay needed support in a day when erroneous theories of the causative factor

were strongly entrenched. Support came from a very young man!

The apt subtitle of this book refers to Dr. Matas as "one of the great pioneers in surgery." Dr. Matas was a pioneer in various undertakings, as in the yellow fever studies just mentioned, though his contributions to surgery were the chief basis of his world-wide fame. One of his early cases stands out as a clear landmark, a "first" in a long record of vascular surgery. The patient had an aneurism of the brachial artery, and in performing the surgery Dr. Matas introduced a novel suture technique in a procedure later called "the Matas operation." This distinct advance was made when he was only about 28 years of age, again illustrating capacities that characterized his long career.

Dr. Matas had close ties with the Tulane School of Medicine. An alumnus, and the most distinguished of all, he served in part concurrently as Demonstrator of Anatomy and Instructor in Surgery from 1885 to 1894, when he was appointed Acting Professor of Surgery. In 1895 he became Professor and Head of the Department of Surgery. On reaching the age of university retirement, in 1927, he was named Emeritus Professor. The library of the School, which held his active interest to the last, has borne his name since 1937: The Rudolph Matas Medical Library.

Dr. Matas was much more than surgeon—scholar of broad interests, humanitarian, a man whose simple ways were a mark of his true greatness. His personal life, like other lives, was not entirely serene. The parents were ill-matched, hence the early family environment was marred. As years passed, his mother showed her true colors as a virago and his sister turned into a vixen—yet he held them in affection. The only child of his marriage was stillborn. For a half-century, Dr. Matas was a "cyclopean," as he termed his state after an eye had to be removed because of an operating-room infection. Trials such as these, along with the happinesses and accomplishments of a long and richly productive life, are set before us in this work.

A bronze portrait plaque in Touro Infirmary was dedicated to Dr. Matas in 1941 as a token of appreciation, in particular for his long service as Chief Surgeon (1906-1935; Emeritus Chief Surgeon, 1935-1957). The plaque carries an inscription: "Your example will remain a tradition and a spiritual legacy to immortalize your fame." The biography is a full and honest recording that will perpetuate the benefits of this example.

HAROLD CUMMINS, PH.D.

Medieval and Renaissance Medicine; by Benjamin Lee Gordon, M. D. Published by Philosophical Library 1959. 843 p. Price, $10.00.

Physicians interested in the historical background of the profession will welcome this book. To compose such a volume is no small undertaking when one considers the period of time it covers and the limited recorded knowledge available about the medicine of these eras. Because of this the book is a somewhat disjointed collection of essays on medicine in various countries, diseases and specialties, most chapters being sprinkled with short biographies of physicians and containing fairly adequate references. In general, the essays are well written and interesting, especially the chapter on the development of university teaching.

However, I have found a few disturbing features about this book. Although a history of medieval and renaissance medicine, many of the essays carry their discussion into the nineteenth and twentieth centuries, (For example, in relation to diabetes there is mention of the recently developed oral hypoglycemic agents). There are many illustrations, mostly portraits of physicians, but there is no correlation between these and the text. In fact, there are two pictures of one Jesse Bennett (plate 41 and plate 52) who is reported to have performed the first Cesarian section in this country. I was unable to find any mention of him in the text, even in the chapter dealing specifically with obstetrics; and, of course, one wonders whether he really belongs to this historical period.

In general, I would consider this volume to be a useful reference for physicians for general interest in medical history, but question whether the serious medical historian will find it of great value.

ALTON OCHSNER, JR., M. D.

Medical Research and the Death Penalty; by Jack Kevorkian, M. D., New York, N. Y., Vantage Press, 1960, pp. 75, price $2.50.

The author of this book believes that criminals who have been condemned to death should be given the opportunity of making their bodies available for medical research experiments under anesthesia, which would terminate in death, as an alternative to being executed in conventional fashion. The arguments in support of his proposal are set forth in the form of a dialogue between the author and an imaginary antagonist. The antagonist is allowed to say all of those things that have been voiced against the proposal to the author in private conversation. The author for his part labors the great advantage to medical science that might be obtained from the experiment, and the fact that the procedure would be a benefit rather than a sacrifice to the prisoner himself.

The author is careful to exclude from discussion all considerations of emotional or aesthetic values, claiming that the matter must be decided on purely logical and factual grounds. He may, however, be making a fundamental error in thinking that it is desirable to exclude emotional

factors from what appears to be a rational decision. It is, perhaps, significant that the author is a pathologist rather than a practicing clinician.

The book is thin, and very readable. Its main purpose is stated to be to stimulate serious discussion about this proposal, but serious discussion is likely to originate only from those who believe in it. I do not think that the book will persuade anybody who starts reading it with a prejudice against the idea of human vivisection.

BRIAN H. MCCRACKEN, M. D.

Adventure to Motherhood; by Offen J. Allan, Miami, Fla., Audio Visual Education Company of America, 1960, unpaged, $2.95.

This is a picture story with supplements intended as an aid in prenatal care. The booklet contains color photographs that take a patient and her husband through the normal prenatal course and delivery. The photographs are interspaced with explanations and helpful prenatal advise.
advice.

The supplements which can only be distributed by the physician, consist of a self-taking obstetrical history and hospital information, notebook leaves, and 23 leaflets dealing with information on a variety of subjects such as the Rh factor, diabetes, cesarian section, constipation, etc. These leaflets are given to the patient as it applies to her. The booklet can be purchased in a bookstore by the patient.

This combination will be a very helpful adjunct in providing prenatal care, especially for primigravidas.

DR. WILLIAM VAN MUYDEN

Acute Pericarditis; by David H. Spodick, Grune and Stratton, 1959. 182 p. $6.50.

An excellent monograph carefully outlined and well illustrated. The discussion of basic anatomy, pathology, and particularly the pathophysiologic changes incident to pericardial effusion are interesting.

The description of the general clinical condition is adequate. The discussion of the individual pericarditides is necessarily sketchy. Generally this is an excellent, basic study of value for all clinicians who encounter this problem.

PAUL T. DECAMP, M. D.

Biopsy Manual; by James D. Hardy, M. D., James C. Griffin, M. D. and Jorge A. Rodriguez, M. D., Philadelphia, W. B. Saunders Company, 1959. pp. 150, Price $6.50.

The authors of *Biopsy Manual* have assembled under one cover a hitherto unavailable review of the techniques used and complications encountered in obtaining biopsy material from practically every organ except those of the central nervous system, the heart, and great vessels.

Dr. Hardy and his co-authors have omitted little in the way of biopsy techniques although subjects which belong in the fields of the surgical subspecialists are necessarily dealt with only briefly. More precise information can readily be located by referring to the carefully selected bibliography. Well written and extensively illustrated, the book is replete with practical and reliable suggestions. Particularly valuable are those dealing with the use and hazards of anesthesia and the complications and errors of biopsy.

Biopsy Manual will be useful to the general practitioner and, most particularly, the surgical trainee. In addition, nurses who are in charge of minor surgery should find it extremely useful.

EDWARD S. LINDSEY, M. D.

Living Beyond Your Heart Attack; by Eugene B. Mozes, Prentice-Hall, Englewood Cliffs, N. J., pp. 212, 1959, $3.50.

A scientific book for the layman who has, or is interested in, coronary artery disease.

It is quite complete and very clear, covering all phases of the subject and quoting many references from medical literature.

However, Dr. Mozes tends to suggest to the reader what he feels is the best explanation or therapy in some situations which are still controversial within the profession.

This would be a good book for a physician to recommend to his patient with coronary disease . . . But the physician should first carefully read the sections on Therapy and Rehabilitation (Chapter 6 through 11) before doing so.

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